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No. 3.—THE NATURAL HISTORY OF VIRUS HEPATITIS OF DOGS (RUBARTH'S DISEASE) *

BY

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As a theme provocative of interest, the above subject held its own well at the Floral Hall, Eastbourne, on the morning of Thursday, September 27th, when Professor George H. Wooldridge, F.R.C.V.S., M.R.I.A. (LONDON), presided over a large attendance, with Miss Lorna H. H. Phillips, of Haslemere, as Recording Secretary.

The CHAIRMAN: The matter we have to discuss to-day is, as you are already aware, The Natural History of Virus Hepatitis of Dogs. Someone's name is applied to it but I do not know if that matters very much.

Mr. Parry is going to give us an introduction to the subject. He may like to make some comments in respect of the paper he has submitted, either amendments or additions, and after that he will give a lantern demonstration. Major Pugh will be able to present his contribution following that, and then the matter will be open for general discussion.

I will now ask Mr. Parry to bring forward his introduction to "Rubarth's Disease."

Mr. H. B. PARRY: Dr. Larin and I feel you have done us a signal honour by asking us to address you on this subject to-day. We are a very young organisation; we have been working on this subject for only a very short time and our experience in the field of distemper-like diseases of the dog is a very short one. To many of you who have had a life-time of experience we feel we are merely novices entering the field. If you would bear that in mind when considering our remarks we should be very grateful.

We also feel that in asking us to give this paper you are in effect not so much honouring us but honouring our Director and our President, who has done so much to help us get this work started in Kennett.

I have been allotted 20 minutes in which briefly to introduce the subject, and in that time I propose to show a series of lantern slides illustrating the salient features and then to give a very brief summary at the end. It is not our purpose this morning to deal in any way with the whole complex of these distemper-like diseases in the dog. We wish merely to place before you certain data which we have been able to collect ourselves regarding virus hepatitis in dogs, or Rubarth's disease, within our own kennels and with a very limited number of outbreaks in the field.

We do not wish to infer in any way that our experience is necessarily representative. We have made no attempt to carry out field surveys, and we are only too confident that the data we have to lay before you will raise more questions that we cannot answer at the moment than questions for which you in practice are looking for answers. In particular, the general biology or ecology of this virus does, we think, make it a rather difficult one on which to offer advice on prophylactic measures.

Mr. Parry then gave his demonstration of slides, and afterwards continued:—

I should like to make one or two final points. First, we are satisfied that the primary aetiological agent is the virus. You can set up the disease artificially. We have set it up in suitable dogs and have produced all the main clinical signs, including the neurological ones, in approximately the same percentage that they are observed with the natural disease. We are not relying merely on the production of a febrile reaction as evidence of the disease.

The next point I want to stress is that in practice you may have mixed syndromes. I should think they would be extraordinarily confusing to diagnose. Then there is the persistence of viable virus for long periods after apparent clinical convalescence, with the appearance of virus in the peripheral blood from time to time. There is the implication of danger, or potential danger, if you are selecting blood donors or serum producers. How important that is we have no means of knowing; all we can say is that it is a potential danger. I might say that that is very much in line with the experience of infective hepatitis in man; precisely the same trouble has been encountered with human serum, particularly mumps convalescent sera, and the importance of infected needles transmitting the disease is well recognised.

We have no conclusive proof that an infected needle has transmitted the disease in the dog; it is extraordinarily difficult to get "cast-iron" proof of that, but we have had one or two cases with a most suggestive history. I should like to stress that the question of sterilisation of needles should perhaps be reconsidered.

The last points, which I am sure have been uppermost in your minds throughout, are the questions "What do you do about the disease and how do you prevent it? What sort of prophylaxis can one recommend?" Well we are very diffident about making any dogmatic statement on these points; we have not done enough work. It is true that the dogs that have had mild experimental hepatitis or inapparent hepatitis are solidly immune to inoculation, but we do not know whether you can get a breakdown of the balance between virus and host with the sudden appearance of clinical disease.

One of the most intriguing problems is that of a kennel of dogs, in which one will suddenly die overnight, and upon examination there are antibodies in the serum in all the

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other dogs, indicating that infection has been in the kennels for months. Why has one dog suddenly become affected fatally? This question makes us extremely diffident about talking of any form of prophylactic vaccination at the present time. We know that some of the American houses are widely pressing prophylactic vaccination. We wonder if they are informed fully of the problems involved.

With regard to the use of anti-serum, it is difficult to produce adequate titres of antibody in the dog, and with the one or two experiments we have done we have had no success with anti-serum in modifying the clinical course of the disease. The whole crux of the position is that the disease is sporadic, and the clinical assessment of the value of prophylactic vaccination or serum-cum-vaccination is going to be difficult and require large numbers for the production of significant data. If you have one dog dying in a kennel it is quite likely that you may not have any more.

Finally, Dr. Larin is very sorry to have been smitten by one of these virus infections (probably influenza) and he asks you to be very kind with me in your questions on serological matters.

Following is the text of the paper, which had been circulated to members in advance:—

Introduction

Distemper has been recognised for two centuries as the main killing disease of dogs and, since the work of Carré, (1905), it has been accepted that the primary aetiological agent is a filter-passing virus assisted in certain cases by secondary bacteria. The studies of Laidlaw & Dunkin (1926-31), followed by those of Dalling (1934), defined one virus which produced an acute fatal disease in ferrets, as well as in susceptible dogs. Rubarth's studies, published in 1947, drew attention to another virus which we feel confident has been, and is, responsible for disease in dogs often confused clinically with Carré's or Laidlaw-Dunkin distemper. This virus does not produce any illness when inoculated into ferrets and it is not easy to reproduce the disease regularly in dogs. Thus distemper and virus hepatitis are caused by two different viruses, the first highly pathogenic for ferrets and the second not pathogenic for ferrets. In our programme of research into the virus diseases of the dog, we have made some observations on virus hepatitis of the dog, which we would like to place before you. This paper should be read as a preliminary report which will raise as many unanswered questions' as those to which it provides an answer, but we hope that it may throw some light on the problems which you meet in practice. In particular we should like to focus your attention on two main points: firstly, the definition of the clinical, pathological and epidemiological features of the disease we know as virus hepatitis of dogs, and, secondly, the importance of the clinically inapparent cases with the continuance of the virus in recovered animals beyond the period of normal convalescence, and the possible rôle of such "carrier" animals in the epidemiology of the disease.

This is not the place to discuss in detail the methods that we have used ; they have been, and are being, published in detail elsewhere (Larin, 1951 a & b; Larin & Parry, 1951; Platt, 1951). The data have been collected in natural and experimental cases of the disease on our own premises and in a small number of outbreaks in other kennels. The development by one of us (N.M.L.) of refinements of the complement fixation reactions for the detection of an antigenic fraction closely associated with the virus, as well as for its antibody, has greatly facilitated our study of the clinically inapparent infections and has provided us with a serological criterion, which, taken in conjunction with clinical and pathological data, has been of great value in establishing a diagnosis in doubtful cases. Since at the moment we have no small laboratory animal in which the the virus will regularly produce severe or fatal disease, it is not always practicable to confirm the diagnosis by reproducing the disease in inoculated animals. However, since the purified antigenic fraction will reproduce the fatal disease on inoculation into suitable pups, we consider that the serological demonstration of the presence of this antigen is, in the light of our present knowledge, strong evidence for the presence of the infection.

The Principal Clinical and Pathological Features

A. Clinical Forms

The disease produced by the virus infection in dogs varies a very great deal and it is essential that we recognise this variability since only a relatively small proportion of cases are sufficiently ill for the owners to draw the attention of their veterinary advisers to them. We recognise four forms of the uncomplicated virus disease, the fatal fulminating, the severe non-fatal, the mild and the clinically inapparent, which have been described in detail in *The Veterinary Record* (Parry, 1950).

(1) *Fatal Fulminating Form*

The dog which has been perfectly healthy and well suddenly collapses and dies within 12 to 24 hours ; a common history is of a dog perfectly well at night found dead in the morning. Dogs of all ages may be affected but it is more frequent in puppies three to nine months old. It is unusual for one to have an opportunity of examing such a case before death, but if this does occur the body temperature is usually 105° F. to 106° F., the pulse is greatly increased in frequency and of small amplitude, narrow and soft. Such dogs will often be alert and active to within two to three hours of their death, when the signs of terminal circulatory collapse develop very quickly, with pallor of mucous membranes and, occasionally, jaundice. At autopsy there may be little obvious macroscopic abnormality. However, there is usually a blood-stained effusion in the peritoneal cavity with a fine, yellow mottling of the liver, which in sections can be shown to be due to a centrilobular necrosis. There is oedematous thickening of the gall-bladder wall. Petechial haemorrhages are infrequent but it is common to find extravasation of blood into the alimentary tract, particularly the small intestine, which in severe cases consists of almost whole blood throughout its length without evidence of any marked discontinuity of the mucosa, although sections show that the surface of the villae has broken down with severe damage to the sub-mucosal blood vessels. If smears of fresh liver be fixed with methyl alcohol and stained with 1 per cent. Giemsa for one hour, about 20 per cent. of liver cells will be found to contain large aggregates of basophilic chromatin in the centre of their nuclei, the so-called nuclear inclusion bodies, which are at least half as large as the nucleus. Large amounts of the antigenic fraction occur in the blood, peritoneal effusion, liver and other organs of such cases.

(2) *The Severe Non-fatal Form*

The dog has a temperature of 105° F. to 106·5° F.,

during the first 24 hours of which no clinical ill-health may be observed and the appetite is but little affected. However, if the cardiovascular system is examined it will be found that there is a tachycardia and the peripheral pulse is diminished in amplitude, narrow and soft. The tonsils are often enlarged and congested, and there may be blood on the thermometer after rectal insertion, though blood-stained stools are rare. In the early stages this type is very similar to the fatal cases, but in our experience if a dog survives 24 hours of the severe disease it nearly always recovers after four to ten days of malaise and reduced food intake, providing there is no secondary bacterial complication of the virus infection. In autopsies of cases which have survived 24 to 36 hours, and are recovering, there is extremely little abnormality that one can detect; smears from the livers of such animals do not show obvious inclusion bodies although in sections centrilobular liver damage can be seen readily. A small proportion of these severe cases shown signs of damage to the central nervous system with motor seizures of varying pattern which are clinically indistinguishable from those usually attributed to so-called " hard-pad " disease; in our limited experience these cases recover completely without treatment in two to three days although in practice they would probably have been destroyed as incurable. In none of our dogs have we seen any delayed neurological sequelae, such as Parkinsonism (chorea) or the syndromes which fall in the category of Perdrau and Pugh's (1930) post-infective encephalitis.

(3) *The Mild Form*

These cases show only slight clinical illness and unless closely examined would not be recognised by the majority of owners. This form is frequently ushered in by hysterical seizures of the running-barking type which may persist for two or three days. During this time the body temperature may be elevated to 103° F., but there is very little change in the food intake, although observant owners may notice slight lethargy or malaise. Close examination of the cardiovascular system shows that there are slight departures from the normal heart frequency and pulse characters but they are not easily detected. In some cases of mild virus hepatitis infection we have observed a peculiar kerato-conjunctivitis affecting either one or both eyes, in which the predominant lesion is a photophobia and a light diffuse opacity of the central portion of the cornea which may involve one eye or both eyes with or without injection of the conjunctival blood vessels. This opacity may persist for 2 to 10 days and often subsides within 24 hours, and it is noteworthy that we have never observed any vascularisation of the cornea, nor has there been any ulceration, while lacrymal discharge is very slight and nearly always serous.

(4) *The Inapparent Form*

In these cases there are no obvious clinical signs of ill-health, but by serological techniques we have demonstrated the development of specific antibodies. Such animals are resistant to subsequent inoculation of virulent virus.

B. Rôle of Hepatitis Virus in Puppy Mortalities

We have observed in some of our brood bitches which have had virus hepatitis during the past 18 months that there has been an increase in the incidence of still-births and neonatal mortalities (by this term we mean that puppies are born alive, that they die during the first week after birth). While it is quite out of the question to deal with the many factors which may be responsible for these neonatal mortalities we would like to report a few of our findings in six recent whelpings, in which 26 of 60 pups born did not survive the first week. In most of these puppies there were no obvious morbid anatomical lesions. Haemolytic and non-haemolytic streptococci have been recovered inconstantly, but the hepatitis virus antigen has been demonstrated in the livers of seven of 14 pups tested, and in four of these seven cases we have had macroscopic signs with peritoneal effusion and petechial haemorrhages. While this work is still incomplete we suspect that hepatitis virus infection, often mild or inapparent, of a bitch can give rise to weak puppies which die during the first week of life, although it is remarkable that not all the puppies in a litter are affected, and the survivors usually thrive normally.

C. Mixed Syndromes

We have had one interesting subsidiary outbreak in our stud kennels six months after the primary hepatitis epidemic, in which 12 four-months-old puppies were involved. Two pups died of acute pleuro-pneumonia, from which streptococci were isolated. Three weeks later, after a damp November week-end of sudden raw cold, two others developed signs of pneumonia, but when the remainder were examined clinically it was found that many of them were suffering from a mild hepatitis syndrome with inflamed tonsils, from which streptococci were isolated readily, and it occurred to us that a streptococcal pleuro-pneumonia might be superimposed upon a non-fatal hepatitis. This was confirmed when four of the most severely affected puppies died. One, without pneumonia, was typical of fatal virus hepatitis, but the three others had severe pleuro-pneumonia with effusion, from which haemolytic streptococci of several types were recovered; careful inspection of the liver and abdominal viscera suggested a moderately severe concomitant hepatitis which was confirmed by demonstration of hepatic damage with only a few nuclear inclusion bodies and by the demonstration of antigen in the liver. This small epidemic occurred during bad weather in winter and we suspect that the primary precipitating factor was exposure, but it served to draw attention to the possibility that bacterial infections, which alone would not be expected to be fatal, will produce a fatal disease if they coincide with a non-fatal hepatitis infection, or possibly " light up " an already existing inapparent hepatitis virus infection. We have had several instances of this sort of syndrome from the field recently—of dogs dying of pleuro-pneumonia with a moderately severe and unsuspected virus hepatitis; of puppies presented with incontinence, which at autopsy were found to have a necrotic cystitis with rupture of the bladder, and a purulent sanguineous peritoneal effusion, which was found to be rich in hepatitis virus antigen, while the liver showed marked lesions of virus hepatitis.

We have recently examined blood from a case of " distemper " in a dog and have found that it contained both Laidlaw-Dunkin virus and hepatitis virus, as well as producing " hard pad " in a ferret. Thus these viruses may well be involved in certain clinical syndromes observed in practice.

Epidemiology

When the disease is first introduced into a community

which has never been exposed, such as the young dogs in our stud unit 18 months ago, the course of the epidemic is extremely rapid and all the animals that became affected had either died or were recovering within a fortnight. This is in marked contrast to our experience of an epidemic of para-distemper, which took nearly five months to pass through a similar kennel of susceptible dogs. The clinical morbidity with virus hepatitis was about 70 per cent., with a mortality in the young dogs of 12 per cent.; four aged greyhound brood bitches from the Greyhound Racing Association showed no evidence of the disease at all. The deaths took place during the first six days of the epidemic, after which no further severe cases occurred.

On two occasions we have introduced new dogs into this stud unit after suitable isolation. The first introduction was of four setters less than one year old ; six weeks later two of these, five-months-old dogs, developed a mild form of the disease while two 12-months-old were not clinically affected. However, since we are not sure that these dogs were not exposed to infection before, it is possible that the older dogs were immune from previous infections. Latterly, we have introduced some of our own older setters recovered from para-distemper and these have shown no clinical illness but have developed a high titre of hepatitis antibodies suggesting recent inapparent infection.

Our experience over the past year in breeding and rearing puppies has also been illuminating. In the summer of 1950 the offspring of the greyhound bitches in the stud at the time of the original epidemic produced puppies which, at three to four months of age, were fully susceptible to inoculation with virulent hepatitis virus. A number of our Irish setters introduced just prior to whelping produced good litters with a low mortality rate and these pups were susceptible to the infection at five months when they developed the mixed hepatitis-pleuro-pneumonia syndrome noted above. The litters born during the last six months have shown an increased puppy mortality rate, to which we have referred, and in many cases the survivors of these litters have now developed specific hepatitis antibody in their blood without having shown any clinical illness. We are thus presented with a picture of virus hepatitis introduced into a clean community producing an epidemic of clinical illness with fatal cases. Later the infection becomes endemic with little or no clinical illness but there is an increase in neonatal mortalities. Clean animals introduced into this environment are likely to suffer clinical illness, and one may expect occasional fatal fulminating cases.

This state of affairs is not peculiar to our kennels ; we have had similar experiences with two outside kennels during the last six months. In one kennel odd sporadic cases of fatal hepatitis, with or without complicating pleuro-pneumonia, have occurred at intervals of some months, while litter mates of affected animals have been apparently normal. In another kennel which we were able to observe closely, the first evidence of the disease was the sudden death of a five-months-old puppy last November. When the remaining 18 animals were examined, it was found that the litter sister of the fatal case had mild clinical illness with no antibody as yet in the blood, but that a number of other young dogs, less than 12 months old, as well as older brood bitches, showed antibodies in their blood, suggesting that they had been exposed to the infection during the past year. We are thus presented with a picture of an infection probably being endemic in this kennel and then for some reason, which we do not understand at present, a fatal fulminating case occurs. Recently a brood bitch which gave a positive antibody test in November whelped a litter of seven to term. One pup died on the second and one on the fourth day, and each showed both serological and pathological evidence of severe virus hepatitis infection. The five survivors are thriving, and are under observation.

Another aspect of the disease we have not been able to investigate very far is the sudden fatal case in a middle-aged dog, a household pet or from a small kennel, in which there is no history of other illness. Why a fulminating fatal disease should develop suddenly in such animals is an intriguing problem.

The Aetiological Agent

1. Some General Considerations

Our data support Rubarth's conception that the aetiological agent primarily responsible is a filter-passing virus. We have reproduced the disease in suitable puppies by injecting intraperitoneally suspensions of liver from animals dying from the disease and from which we could not demonstrate any bacteria or protozoa. The clinical, pathological and serological features of the natural disease have been reproduced in inoculated animals and each major sign has occurred in approximately the same percentage of animals as in the natural disease, *i.e.*, we have reproduced all the principal features of the natural disease as we have observed it in the field. Our animals have been in a good state of nutriture, have carried a moderate to light roundworm infestation, and, as far as we have been able to ascertain, have not been affected with coccidia (the counting method would reveal oocysts if present in excess of 100 per gram of faeces). Rubarth draws attention to the fact that some fatal cases of the disease carry a heavy roundworm infestation; we have noted this in one or two instances, but in our experience it has been an inconstant finding and the rôle of alimentary helminthiasis in influencing the severity of the disease remains to be established. We have cultivated the aetiological agent in embryonated hen's eggs and material from the 12th egg passage has reproduced the disease in puppies. The infective agent will pass through a gradocol membrane of 750 mμ A.P.D. (Miles, Parry, Larin & Platt, 1951).

We have examined four strains of the virus—two English strains, including our own, one from Professor Rubarth and one from Australia. They are antigenically similar in the complement fixation test, and on methanol precipitation in the cold an antigenic fraction can be prepared which when resuspended in normal saline yields a clear fluid capable of setting up the disease when inoculated into dogs (Larin, 1951a).

After the inoculation into susceptible dogs of either one of the four strains of virus, or of the methanol precipitated fraction prepared from them, the dogs develop specific antibodies in their sera, which can be detected in the complement fixation and ring precipitin tests. Complement fixing antibody usually appears 3 to 10 weeks after the primary infection, reaches a certain titre and then wanes after 2 to 6 months ; it is barely detectable after 6 to 12 months. However, dogs which have lost their circulating complement fixing antibody have been fully immune on challenge inoculation with virus.

2. Ecology of the Virus in the Dog

After intraperitoneal inoculation, antigen was detected

in the peripheral blood within 3 to 8 hours in considerable quantities. It disappeared at the height of the hyperthermic reaction 48 to 72 hours after injection, to reappear again at 60 to 80 hours, after the temperature had begun to fall. This reappearance of antigen was followed by a second rise of temperature at 80 to 96 hours, after which the temperature returned to normal by about 120 hours, when no antigen was detected in the peripheral blood. Thereafter antigen may reappear in the peripheral blood, even after full clinical convalescence. We have detected antigen in inoculated dogs on several occasions 6 and 8 weeks after apparently complete recovery. We have also injected the suspension of the liver from a dog which died suddenly three months after clinical recovery, and whole blood from natural cases six months after clinical recovery, and have set up with both inocula inapparent infections in young dogs with the development of serum antibodies and complete resistance to subsequent challenge inoculation with virus. Thus we suggest that the virus may persist for considerable periods in the clinically recovered animal, and that viraemic episodes occur from time to time without obvious signs of ill-health.

The importance of this observation when selecting blood donors or serum producers needs no further stressing. It is in line with the experience in human medicine, where the risk of transmitting human infectious hepatitis by blood transfusions or inadequately sterilised needles used for injections is well recognised. Whether in the dog such viraemic phases are normally associated with recrudescences of clinical ill-health we do not know, but it is most suggestive that we have observed several recrudescences of the kerato-conjunctivitis syndrome several weeks after the primary infection and recovery. We have not been successful in demonstrating antigen in the blood of these cases at the time clinical eye signs are observed. We have also noted a recrudescence of the kerato-conjunctivitis syndrome when a recovered dog has received a challenge inoculation of virulent virus to test its resistance.

The rôle of persistence of virus in disturbances of the reproductive cycle in dogs is under investigation at present. Two facts are worth noting: firstly, that several peracute cases of the disease have occurred in bitches which have just come into oestrus, and, secondly, the high incidence of neonatal mortalities in litters born of dams recently recovered from the disease and the demonstration of antigen in some of these puppies prior to suckling. This suggests that the virus may pass the placental barrier into the foetus.

3. Ecology of the Virus Outside the Dog

The course of our own epidemic, and also our experience in several outside kennels, strongly suggest that the infection continues on premises for some time, possibly years. This, in our experience, is very different from infections with viruses of the distemper complex, which in a closed community tend to be self-limiting; we have had no reappearance of this latter infection in our premises, although these have not been treated differently from those in which the virus hepatitis epidemic occurred. Whether this persistence of the hepatitis infection on premises is due to excretion of virus by recovered "carriers" or is due to the survival of viable virus in contaminated kennels is not yet known. We suspect that both processes may occur since the hepatitis virus is much more resistant to heating and desiccation than the Laidlaw-Dunkin virus. Hepatitis virus retains its ability to set up clinical disease after heating to 56° C., is stable at −20° C. for many months and will withstand repeated thawing and refreezing. Thus it seems highly probable that the virus will remain viable outside a host for a considerable time, although we have not yet been able to establish this experimentally.

The rôle of arthropod vectors in the transmission of the disease is of some interest. We have detected antigen in lice collected from two sick dogs, and have set up a very mild infection in a dog with inoculations of suspensions of such lice with the subsequent development of specific antibody in the dog's serum. It is probable that lice may play a part in transmitting the disease under natural conditions, although we have had no opportunity of attempting to transmit the disease by feeding lice engorged on dogs in the viraemic phase of the disease and then placing them on susceptible animals.

Whether there are carriers of the virus other than the dog (and probably the fox) under natural conditions we do not know, but under experimental conditions we have had some evidence that the virus can multiply in the cat; usually, however, the infection is inapparent.

4. The Nature of the Immunity

One of the most striking features of the disease is the variability of the response of apparently similar dogs to the infection under both natural and experimental conditions. Why this should be so is under investigation. As we have mentioned above, the immunity of a recovered dog to inoculation of virulent virus cannot be explained satisfactorily on the basis of serum antibody levels. Even when the titre of antibody is very low the dog is immune and remains so even when the serum antibody titre has fallen to undetectable levels after six to 12 months.

It seems not unlikely that the dog's enhanced immunity to further infection may be due in part to the virus harboured in some organs of the animal, but that the cellular damage that ensues is not enough to produce clinical signs of ill-health. It is difficult to explain the occurrence of antigen in puppies dying within 48 hours of birth unless one postulates that the bitch has continued to harbour the virus. It is also of interest that exposure has precipitated an acute pleuro-pneumonic syndrome in dogs probably already harbouring the virus, with death from primary streptococcal pneumonia, but with secondary hepatitis. We are, therefore, coming to think that latent infections with the virus may be common and that the immunity following exposure to hepatitis virus may depend in part on the continued presence of virus in the host, namely, on a state of premunition, and that this state may be broken down by unusual stresses such as exposure or concomitant bacterial or helminthic infections, which are not of themselves serious.

If further studies show these ideas to be correct, the control of the disease by prophylactic vaccination raises some problems, which should be approached with caution.

Applications to Veterinary Practice

1. Diagnosis

There are five criteria which we apply in establishing a complete diagnosis, namely, the clinical syndrome, the morbid anatomy, the histo-pathology, serological tests and animal inoculations.

We should distinguish two kinds of diagnosis—diagnosis of the infection and diagnosis of the disease. *Diagnosis of the infection* has been facilitated greatly by the new serological tests. Antigen can be demonstrated regularly in the liver, peritoneal fluid and other tissues of animals dying of the disease; in sick animals the antigen is present in the peripheral blood from time to time, while a

retrospective diagnosis can be made by demonstration of antibody over a period of two weeks to 6 to 12 months after the primary infection. *Diagnosis of the disease* depends upon a careful appraisal of the clinical and pathological evidence, as well as demonstration of the infection. Hepatitis virus probably affects in the main the permeability of blood vessels, causing extravascular loss of fluid and blood cells with death due to oligaemic shock; only occasionally does clinical evidence of hepatic damage with jaundice occur. In the uncomplicated disease the clinical signs should conform to this pathologic physiology. Liver function tests and the examination of liver biopsy specimens may prove of value, but at present their worth remains to be established.

The fatal fulminating case can be detected usually on the morbid anatomy alone with confirmation by the finding of nuclear inclusion bodies in liver smears. In the past such cases have been attributed to acute poisoning or peritonitis because there is often no clear-cut evidence of any contact with a known diseased dog. Where jaundice is also present it may be confused with leptospirosis, but in the latter disease there is typically no oedema of the gall-bladder wall, nor are inclusion bodies present in the liver cell nuclei, while the clinical course of the disease is usually longer. Serological examination of liver and the peritoneal effusion for hepatitis virus antigen is essential when the diagnosis is in doubt.

The severe non-fatal case may provide some difficulty since the principal clinical signs are hyperthermia with only moderate malaise, etc.; in fact, the most striking feature is the severity of the hyperthermia without signs of systemic illness affecting appetite, etc. However, the cardiovascular signs provide valuable indications. The serological examination for antigen of single samples of peripheral blood from these cases is of limited value owing to the short duration of the viraemic phases. A positive result is most useful, but a negative one does not necessarily exclude the disease. Should such cases die when apparently recovering, or in the very early stages of the disease, the morbid anatomy may show no significant departures from normal and in liver smears very few inclusion bodies can be found. Such cases are extremely difficult to diagnose with certainty without repeated serological tests for antigen. Well-developed intranuclear inclusion bodies are present during only a relatively short phase of the disease process, probably lasting four to 12 hours, and if the animal dies outside this phase the demonstration of inclusion bodies is not easy, for they must be carefully differentiated from *post-mortem* karryorrhectic changes with clumping of the nuclear chromatin.

The mild cases usually do not come to one's attention, but sudden outbreaks of hysteria which it is not possible to attribute to other causes may be viewed with suspicion. The occurrence of the kerato-conjunctivitis syndrome we have mentioned above is almost diagnostic. A retrospective diagnosis can be made by demonstrating the development of antibody.

The inapparent infection, of course, is only diagnosable by laboratory means, either by demonstrating virus or antigen in a fortunately timed blood sample or liver biopsy specimen, or, retrospectively, on the development of antibody in the peripheral blood.

2. Treatment

The treatment of affected animals is, at the present time, largely a matter of good nursing. Keep the animals in a warm place on a light, easily digested diet high in carbohydrate with a good protein and a moderate fat content. Fluid intake should not be forced and the parenteral administration of fluid can be dangerous. If the circulating blood volume is low, transfusions of whole blood, plasma or blood substitutes should be given, selecting a donor which has recovered from virus hepatitis. The value of hyperimmune serum in modifying favourably the course of the disease remains to be established. We have not had any opportunity of assessing the value of the newer antibiotics, but aureomycin has proved of little value in the treatment of infectious hepatitis of man.

3. Control

The control of virus hepatitis raises problems which are more complex than those encountered with Laidlaw-Dunkin distemper, since latent infections and prolonged persistence of the virus outside the body have not been reported in that disease.

(1) *Active Immunisation*

If the immunity of the recovered animal depends on the maintenance of a state of premunition with persistence of viable virus, vaccination is not a simple matter. The use of killed and living vaccines raises a number of questions, which we are investigating now, namely, the most suitable strain of virus to use, the best age to vaccinate, the risk of producing permanent "carrier" animals, in what circumstances the state of premunition can be disturbed with recrudescences of clinical illness, and what are the risks of introducing hepatitis virus up and down the country. Until we know more about these questions, until the distribution of virus hepatitis throughout the country and the incidence of severe illness associated with the virus has been determined, we feel it is unwise to advocate any programme of widespread prophylactic vaccination.

(2) *Passive Immunisation*

The use of hyperimmune serum for the protection of in-contact animals might appear to be of value, but at the moment it is an open question. Two considerations arise. Firstly, at present the only practicable way of assessing the potency of such a serum is on its complement fixing antibody titre and the production of sera of high antibody titre is not easy; furthermore, such sera may contain virus. Secondly, only a very small percentage of in-contact animals are likely to develop the disease in any event and there is no way of detecting these at present; in fact, in instances where a single animal has died, most of the others have been found to have antibodies indicating recent infection and have shown no ill-health. The evaluation of a serum in a disease as sporadic in incidence as virus hepatitis in the dog requires field trials on a very large scale, and these have not been reported yet in this country.

(3) *Hygienic Measures*

As infection is probably conveyed principally by the ingestion of material contaminated with faeces and urine containing the virus, every effort should be made to break this cycle by strict cleanliness in kennels and by thorough cleaning of food utensils, hands, etc The hepatitis virus is more resistant to heat and drying than the Laidlaw-Dunkin virus, so the best means of attack is to remove as much virus as possible by cleansing and washing down, unless it is practicable to use a blowlamp.

The risk of conveying the infection in contaminated biological products, such as sera and vaccines produced in dogs, is a very real one. Sera against distemper can contain the hepatitis virus antigen and there is at least one report in the literature of acute fatal jaundice in dogs following the use of anti-distemper serum. In the light of our present knowledge this could well have been due to contamination of the serum with hepatitis virus. Whether the infection can be transmitted by inadequately sterilised needles, we do not know, but recently we have had a case which might have been due to this cause ; it would be a wise precaution to pay particular attention to the sterilisation of needles and syringes used for injections in canine practice.

Summary

Virus hepatitis of dogs is a disease which may be confused with Carré's or Laidlaw-Dunkin distemper. Severe forms of the disease show disturbances of the circulation simulating oligaemic shock, with, occasionally, jaundice. The mortality rate is low, but the consequences of the infection can be serious. Fatal cases exhibit extravasations of serous fluid and whole blood into the peritoneal cavity and abdominal tract, with centrilobular necrosis of the liver, but usually no jaundice. Mild and clinically inapparent infections are common, and would pass unrecognised in the field. The disease once introduced into a kennel tends to become endemic with a low clinical morbidity rate, but with possibly some increase of neonatal puppy mortalities. Mixed infections with bacteria and of the distemper and complex viruses can occur.

The disease is due to a virus distinct from the distemper group, and is not pathogenic for ferrets. We have reproduced in dogs the various clinical manifestations of the natural disease and in approximately the same percentage of cases with inocula free from bacteria and protozoa. Some cases show neurological signs indistinguishable from those seen in para-distemper. The virus can be cultivated in embryonated hen's eggs.

The development of an improved complement fixation test for an antigen closely associated with the virus and for antibodies in serum has allowed us to follow the course of the infection more accurately. Clinically inapparent infections are common, and the virus reappears from time to time in the peripheral blood of fully recovered animals. Recovered dogs are solidly immune for at least a year and this is independent of complement fixing serum antibody titres. Immunity may depend on a state of premunition, in which the animal continues to harbour the virus.

The virus is relatively resistant to heat and desiccation and the infection remains on infected premises for some months. The diagnosis and various matters relating to the control of the disease by prophylactic vaccination are discussed.

The danger of transmitting the infection by the use of biological products containing the virus is stressed.

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The Chairman: We have listened to and seen a very intriguing account of this disease, about which I for one know little or nothing. It conjures up in my mind quite a number of the pictures one saw in earlier days during outbreaks of infections resembling distemper, which many years ago I suggested we should group together not under the name of " distemper " but " *the distempers* " of the dog.

I do not propose to enter into this discussion at all, because if Mr. Parry is unable to give us more precise or in any way dogmatic information on certain aspects it does not behove me to do anything of the kind.

We regret that Dr. Larin is unable to be here, and I am sure I express the wish of all when I say we hope he will make a speedy recovery. We ask Mr. Parry to give our sympathetic feelings to Dr. Larin when the opportunity arises. (Applause.)

Without further ado I will ask Professor Pugh to take on the difficult task of opening the discussion and giving us further information on this elusive disease.

Discussion

Professor L. P. Pugh, B.Sc., F.R.C.V.S. (Cambridge): I am sensitive of the privilege of being asked by the " National " to open the discussion on this paper, the more so because I was privileged to perform a similar duty on Dr. Trevan, Montgomerie and McIntyre's paper on so-called hard pad disease at the Southport Congress. The study of the complex clinical picture that passed for so many years as canine distemper has always intrigued me and although I am afraid I have not contributed much to its elucidation, I have at least subscribed fairly consistently over the last 15 to 20 years to the idea that other viruses besides the Laidlaw and Dunkin virus must be at work if one was to explain adequately all the clinical phenomena met with. It was therefore with great interest and satisfaction that I studied Parry and Larin's present contribution to this subject—a contribution which I will say forthwith is, in my opinion, also an outstanding one.

I presume the authors would be the first to admit of their good fortune in that this disease should have broken out accidentally in one of their kennels known to be free of both Laidlaw and Dunkin and hard pad virus and at a time when they were reasonably well prepared to deal with such an occurrence—but I am equally sure you will agree with me that they are to be congratulated on the thoroughness with which they took advantage of this accident. It was Charles Nicolle, I think, who said " Chance favours only those who court her." The result is that we have presented to us to-day for the first time a really clear-cut clinical picture of Rubarth's disease as occurring in a closed community under conditions that more or less excluded the possibility of its being mixed up with any of the other common canine infections.

The authors' division of the clinical manifestations of the disease into four main forms appears adequate and easy to follow.

(1) *The Hyper-acute Fulminating Form.*—We must all have seen this type of case and during our careers possibly have tried to hide our ignorance of the pathology of the condition behind some such

diagnosis as that of acute meat poisoning and, as these cases usually occur singly, have probably got away with it. It is fortunate that in these cases a rapid diagnosis can usually now be made by touch preparations of fresh liver tissue, and that one does not have to wait for sections to be cut.

(2) *The Severe Non-Fatal Form* that recovers more or less spontaneously, appears to me to be the ideal type of case to enhance a practitioner's reputation with his clients. Although the authors state that spontaneous recoveries appear to be the rule in a closed community during a recent outbreak, yet I am strongly of the opinion that in the field, more especially in an area where an epizootic is spending itself out, one will find many cases showing secondary bacterial complications where recovery is by no means spontaneous, and it is in these circumstances that a clinical differentiation between true distemper, virus hepatitis and "hard pad" disease becomes in my experience most difficult. In regard to the kerato-conjunctivitis which the authors mention in this form of the disease and which they appear to consider almost pathognomic of Rubarth's disease, I would say that I have seen quite a number of cases of leptospirosis with this complication. It is, of course, arguable by the authors that the leptospirae had stirred up a latent Rubarth's infection.

(3) and (4) *The Mild and the Clinically Inapparent Forms* are from the practitioner's point of view of the very greatest interest and importance. The suggestion that the virus may persist for a considerable time in clinically recovered animals and that periodically the virus may come out and appear in the blood stream, rendering the dog dangerous to others, is one that is both serious and fraught with all sorts of legal possibilities for those in practice. More work to verify such a suggestion obviously is needed—especially as this "carrier" state is bound up with what one might call a rather fragile type of immunity, and one that is not yet well understood.

Although the authors have been careful to observe in their paper that there are many possible factors which may be responsible for neo-natal mortalities, they produce some suggestive evidence to incriminate Rubarth's virus as a possible cause of some of these fatalities—the bitch again acting as the "carrier." This also is a most important suggestion and is one that will certainly be followed up by other workers in this field.

The authors state that the infection is probably conveyed by the faecal-oral route rather than by droplet infection. In this connection there was an interesting suggestion recorded as recently as June this year by Poppensiek, G. C., and Baker, J. A. (*Proc. Soc. exp. Biol. Med.* **77.** No. 2. June, 1951), that urine is important in the spread of the disease, in view of their finding virus in the urine in spite of its absence in blood, saliva, nasal washings and faeces.

There is a point on which I should welcome further information. Parry and Larin state, "Well developed intranuclear inclusion bodies are present during only a relatively short phase of the disease process, probably lasting 4 to 12 hours and, if the animal dies outside this phase, the demonstration of the inclusion bodies is not easy." I had certainly not gathered this impression from the literature—in fact, I thought that pathologists generally seemed to rely rather largely on the finding of these inclusion bodies for the diagnosis of the disease. Is it a correct assumption on my part, therefore, to think that the writers' experience has led them to conclude that a negative finding is by no means conclusive, in fact it may even be of common occurrence, and that they consider their modified complement-fixation test to be the real answer from the diagnostic point-of-view?

In view of Rubarth's suggestion (1947) concerning the similarity of his virus to Green's fox encephalitis virus and also of the work of H. A. Siedentopf and W. E. Carlson (1949) (*J. Amer. vet. med. Assoc.* **115.** 109) in which they claim to have shown that the two viruses have common antigenic properties and should be considered identical—it appears to be highly desirable that the authors should at an early date compare their strain of virus with Green's fox virus, using their complement fixation method. If they are able to confirm the identity of the two viruses (and I think they will), then much useful information could surely be gleaned from the large amount of work carried out and published in the States by Green and his co-workers between 1930 to 1947.* It might be of some interest to note in this connection that in a paper published in *The Veterinary Record* (June, 1937)† I used these words: "As a result of these clinical observations it has become more and more evident that in all probability there exists among dogs in this country more than one disease hitherto described as 'distemper' and that one of these diseases is in many of its characteristics very similar to, or even identical with, the disease described in America as fox encephalitis." Holding these views, I sent material from a number of cases for R. G. Green's examination and criticism. In his reply, dated June 26th, 1937, he said: "After further study of all the material you have sent me, I am still of the opinion that fox encephalitis and 'dry' distemper are distinct disease entities. We now recognise not only inclusion bodies for fox encephalitis but also specific inclusions for distemper in foxes, the virus here being identical with that of Laidlaw & Dunkin. These inclusions are cytoplasmic bodies found commonly within epithelial cells of the bronchi, in the bladder, and in the bile ducts. They are identical with the cytoplasmic bodies recently described by de Monbreun in the *Amer. J. of Pathology* (12 pp. 187-212, 1937). This author has mixed things up terribly by finding fox encephalitis inclusions in dogs and including them as a part of the distemper picture." These references to the past mean little more than that one did at least consider, as far back as 1935, that Green's virus was probably responsible for some of the clinical picture of canine distemper as occurring in this country. Green's negative findings with my material might be explained by the fact that I had purposely selected the same from well-established cases, the very type of case in which Parry and Larin now state they have difficulty in showing inclusion bodies. My own view is that I was probably mixing up clinically the fox encephalitis syndrome with the hard pad syndrome, the latter, of course, not having then been described, and that I had selected material from cases showing focal nervous phenomena. It has been said that veterinary diagnosis is often "a shrewd guess based on experience." The value of Parry and Larin's work is that much of which has hitherto been guesswork seems now established on a more factual basis.

As such, their paper, in my opinion, not only constitutes another very important landmark in the elucidation of the old canine distemper syndrome in this country, but is also at the same time a contribution to pathology that may well have important bearings on infectious hepatitis in man.

The Chairman: I now declare the meeting open for general discussion, and I hope that a number of you will come forward and try to explain some of your problems in the hope that Mr. Parry or Professor Pugh will be able to help you in some solution of them. I myself am so far bewildered and I should want a fair amount of time to collate the impressions I have gained. But we should be delighted to hear of some of your experiences of this disease and of the conclusions you have come to based on those experiences. Mr. Parry is a brave man and has undertaken to attempt to answer some of your conundrums.

General Discussion

Mr. J. K. Bateman (Northaw, Middlesex): I join in paying tribute to the paper presented by the joint authors, Messrs. Parry and Larin, and to the admirable opening paper by Professor Pugh.

Looking back over the last 25 years or so one is struck by the great attraction pathologists, research workers and others have had towards the canine distemper syndrome. Most of the work has been directed towards finding the cause, blame or credit for the symptoms exhibited. Bronchi-septicus disease, brown mouth disease, haemolytic streptococcal disease, Carré virus, hard pad virus and now Rubarth's hepatitis virus have all been reported and in greater or less degree investigated as being either responsible for all or part of the train of symptoms associated with the disease.

One thing is certain, in my opinion, and that is that the original disease is still with us, so we need not fear that we shall run short of material at present. Another thing is equally clear and that is that the work of Laidlaw and Dunkin followed by that of Dalling and others led to a vaccination procedure which was able to cut out completely the whole train of symptoms in a

* Green, R. G., *et al. Amer. J. Hyg.* 1930-1947.

† Pugh, L. P. (1937.) *Vet. Rec.* **49.** 757-761.

very high percentage of cases and most certainly in those which showed a rise of temperature four to six days after the virus injection.

My experience with greyhounds is that the vaccination process to-day only very seldom immunises against the original disease and that I very often see the whole syndrome in groups of young dogs which have already been vaccinated.

This state of affairs led Montgomerie, McIntyre and Trevan to investigate and to report on the hard pad virus and it is not surprising then that Parry and Larin should have followed up Green and Rubarth in investigating viral hepatitis which is said to produce a distemper-like disease.

I appreciate that to-day's paper is in the nature of an interim report and I sincerely hope that it will not be too long before we are given further instalments. I very much regret that we are not given any indication of the scale of the experiments, nor of the number and nature of the controls. We must look forward to that.

With some of the work I am in agreement. As one who has just over 100 brood bitches whelping almost as often as they can be successfully mated, I am very much alive to a new condition which has appeared in the newly born during recent years. The puppies are sometimes stillborn, but more often they are whelped to time and some are so lacking in virility that they cannot suckle. The majority, however, are puppies which feed, remain dreadfully unthrifty and have a greyish, loose, sticky motion. The disease is not a killer in this type, but merely renders the puppies of such low calibre as to be nearly useless for our purposes. After some weeks they eventually begin to recover. I was well aware as a clinician that this was not distemper, because recovered puppies were not immune to clinical distemper. The brood bitches are in every case distemper immune and though there are always 60 to 70 puppies on the premises there has been no case of clinical distemper on the premises for some nine years. I have to record that no samples or specimens have gone to any laboratory, but I can very well see how this type of malady could fit into the picture presented to-day by Messrs. Parry and Larin.

I, then, agree that there is a condition, new to me, which has arisen in the last seven years visibly affecting young puppies in a manner similar to that described. Further, I have been able to eliminate this disease by treatment of the in-whelp and nursing mothers. This would suggest, then, that this new pathogen exists in the adult dam.

Those same pups vaccinated at five weeks against *Leptospira ictero-haemorrhagiae* and later against canine distemper are still susceptible to the canine distemper syndrome which all of us are so anxious to determine and against which Laidlaw and Dunkin were able to vaccinate successfully.

With regard to to-day's paper and the results attributed to the Rubarth virus in the *adult* dog I am not at all happy. It may be that I shall have reason to change my views, but at present I feel that the authors have been over enthusiastic in attributing the deaths of the adult dogs to Rubarth virus. Their premises are not convincing.

Particularly would I maintain from my experience that if the disease does exist in the adult it does so only in a very limited degree. We do have an occasional case of haemorrhagic enteritis and may have a case of jaundice in a young dog of unknown history, but our death-rate from gastro-enteritic diseases other than clinical distemper in the mature racing dogs is only small and not above 1·5 per thousand.

But I am one who tries to immunise these young jaundice- and distemper-vaccinated pups and to expose them to clinical distemper infection, prior to training for racing, thanks to the benevolence of the G.R.A. For some months I have collected batches of these at about 12 to 15 months old and have injected them with febrile blood subcutaneously. That is, I have withdrawn blood taken from a clinical distemper case whose temperature was 104° or over. I have injected small doses of this blood into my saplings, have had severe reactions and in nearly all such cases have had absolutely typical diphasic temperature charts and distemper pictures. One out of 57 had developed the nervous type of disease and was destroyed. Tonsillitis is common but hysteria has never been seen; in fact, there has been no hysteria in our premises since 1942. All other dogs have recovered.

From one such case, "Ice Mermaid," which reacted at five days and which had only recently been vaccinated against distemper I collected 10 c.c. of blood and took it to Messrs. Parry and Larin with the following results.

(1) Serological examination by the complement fixation test gave strongly positive results for both antigen and antibody of canine hepatitis virus.

(2) Whole blood was inoculated subcutaneously into four six to eight months old dogs, two mongrels which have had inapparent natural infections of virus hepatitis and two greyhounds recovered from severe experimental virus hepatitis and two normal ferrets from our stud. The dogs received 0·25 ml. and the ferrets 0·1 ml.

All the dogs showed a mild febrile reaction from the seventh to the tenth day, with temperatures of 102° to 104° but without clinical signs of ill health. On the 15th day one greyhound became definitely ill with diarrhoea and died on the 16th day. Autopsy of this dog showed recent large haemorrhages into the lungs and little else apart from old adhesions of the liver to the diaphragm and around the gall bladder, which we attributed to the previous virus hepatitis. The three surviving dogs then showed fluctuating temperatures, between 101° and 103°, but without any malaise or reduced food intake until the 22nd day when the second greyhound showed a sudden rise of temperature to 104° from 101°; it died on the 23rd day with a severe broncho-pneumonia of both lungs and pleuritic adhesions. The liver was also adhesed to the diaphragm. The two mongrels have continued to show some fluctuation of temperature, between 101° and 103·5°, but have showed no clinical illness and were destroyed on the 34th day; the autopsies revealed nothing abnormal.

Both ferrets showed signs of illness from the 11th day onwards with erythema of the nose and the development of vesicles. One ferret was killed on the 17th day to harvest virus, when *in extremis* with many vesicles around the mouth and chin. Ferret No. 2 by this time, namely, the 17th day, had apparently fully recovered and was feeding well; after about the 30th day the pads began to become thickened and on the 34th day they were very markedly thickened and were peeling over the middle portion—the ferret had also become unwell again and it was killed, *in extremis*, on the 34th day and the virus harvested.

Comment.—These studies would indicate that the strain or strains of viruses in the sample from "Ice Mermaid" contained a ferret-pathogenic virus which produced both the clinical Laidlaw-Dunkin disease in one ferret and so-called "hard pad" in the second ferret. This is the first time that we have succeeded in producing so-called "hard pad" in our ferrets and it is perhaps of interest that this ferret was ill at the same time as the first ferret but survived and it may be that the hyperkeratosis of the pads represents a more chronic form of distemper in the ferret.

Of the dogs, all of which should be fully susceptible to Laidlaw-Dunkin distemper, the two mongrels were affected only slightly, while the two greyhounds were much more severely affected; the death of the first one on the 16th day was probably due to virus infections alone, while the second dog died of superimposed bacterial broncho-pneumonia. We are a little uncertain as to what rôle concomitant virus hepatitis might have played in the death of these dogs, but it is likely that their deaths were in part due to co-existing hepatitis; the microscopic sections should help us to evaluate this.

But what was the donor's past and subsequent history? It was a 14 months old bitch, one of a litter of four Leptospira-immune saplings. These four saplings and other five of similar age and history had been vaccinated against distemper (Vaccine Virus B.W.) on May 19th and May 28th of this year. There was no temperature reaction up to the 11th day and on that day, June 8th, I gave each of the nine saplings 0·2 c.c. blood from a donor dog (104·6° of the previous day). Every one reacted by the fifth day and by July 1st—23 days after live blood injection—all had recovered from an apparent attack of clinical distemper with the usual symptoms of nasal discharge, diarrhoea, etc. They are still fit and well.

One is tempted to ask why I should have this wonderful result when febrile blood from one of the batch caused such havoc at Kennett. Perhaps Messrs. Parry and Larin can throw some light on this. Meantime it must be noted that all of the batch were Leptospira immune. It would seem that since the blood was fatal to ferrets, the distemper vaccine virus given earlier had not conferred an immunity to Laidlaw-Dunkin virus.

It does seem, too, that this hepatitis virus is connected in some way with clinical distemper. We can leave the elucidation of the problem to the principal speakers.

Before I close I would make one more remark. It is this. I have seen no evidence whatever, in the course of my distemper blood injections, that I have transmitted any other virus. Those acquainted with the handling of the Carré virus will know how easily it can die *in vitro* even at 0° to 20° C. I have had that experience several times and injected distemper-susceptible dogs with blood in which the Carré virus no longer existed, but in which presumably the hepatitis virus could still exist. I am not in a position to do serological tests but even repeated and various similar blood injections have failed to produce any clinically apparent illness in the still distemper-susceptible dog.

Further, the greyhound bitches sent by me to Messrs. Parry and Larin and mentioned in the paper were guaranteed distemper

immune. Most naturally I wonder what the significance of that may be in the assessment of their resistance to the hepatitis illness. One was 22 months old when sent to Kennett and one four years old.

Might there be something at the receiving end of the injection? Sway-back is a disease which I believe can be eliminated by the addition of certain supplements to the food. Why is there a different reaction when we inject live virus into dogs? Is there something at the receiving end in the individual animal to determine whether or not its central nervous system will be affected? Or something at the receiving end in the liver to determine if it will have inclusion bodies in its liver at that time?

For instance, Mr. Parry, I believe, feeds his dogs on whale-meat. What do the whales feed on? A dog fed on cow meat will have in its system those factors on which the cow fed. Obviously, according to the mineral and other content of the various pastures the cow meat will vary. It may be that there is an X factor present in some samples which, if determined, can supply the answer to my question.

Dr. J. T. Edwards, D.Sc. (London): I feel it my duty to come along at this stage of the discussion to set things—I hope—right! (Laughter.) We are grateful to Mr. Parry for having brought before this meeting a very full and fair account of a new disease to the British veterinary profession. But I do not think he has sufficiently stressed the fact that we owe the greater part of the information which we now have to the young Swede Rubarth, who a few years ago, after a very complete and careful study, described this disease, which is as different from distemper (or anything else we knew of) as chalk is from cheese.

Mr. Bateman, the last speaker, has confused the issue, I submit to him with the greatest possible respect for his ability as a clinician, by describing hard pad disease and all sorts of odds and ends which have absolutely nothing whatever to do with Rubarth's disease. Rubarth's disease is as similar to distemper as typhoid is to tuberculosis, that is to say if you parallel the virus field with the bacterial field. We know there are now probably more viruses prevalent in the world than there are bacteria.

Too much and too little has been made of the work done in this country on distemper. We were too much inclined to believe that the work of Laidlaw and Dunkin was complete. They deserve recognition and every credit for having published a few papers which were excellent at the time in their limited way. But I do not think that sufficient credit was ever given to the French veterinary surgeon Carré for having placed it on record quite distinctly several years previously that distemper was caused by a virus. We got confused for several years afterwards in this country and America through assuming that certain secondary bacterial invaders were the primary cause. Laidlaw and Dunkin confirmed and extended Carré's work, yet there remained much more to do in the way of studying the infection and the difficult work of devising safe and effective measures applicable to general practice; Dalling was responsible for much of this.

What do we know about this new disease, Rubarth's disease? We know nothing about the cure. We know, too, nothing about prevention. And I am very glad that Mr. Parry was never tempted to embark on that aspect before he had studied the disease itself thoroughly and knew more about it.

With regard to its natural history, I wish he had told us more, because it is in that respect that the disease is so curious. It is utterly unlike distemper. That is made evident by the odd case which dies so very suddenly though the symptoms have hardly been seen at all. The dog is just found dead, but upon *post-mortem* examination there are lesions which are characteristic. The disease cannot be mistaken for anything else, so well did Rubarth originally describe the lesions which are to be found in this disease.

Although the disease is made evident and manifest by these occasional sudden deaths and the characteristic *post-mortem* lesions, we know now that there are all grades of infection varying in severity from the fulminating disease to the kind of disease which is clinically inapparent.

Moreover, we do not know how the disease is transmitted. It is not easily transmitted by contact.

But there are other diseases we know very well which are also not easily transmitted by contact: or, at any rate, the conditions under which they are naturally transmitted cannot readily be discerned and imitated, artificially, in the laboratory.

In the course of discussion at a meeting of the "Central" held at the Royal Veterinary College, London, several months ago, when Mr. Pay presented a paper on Rubarth's disease, I suggested that in the same way as, in cattle, canine distemper runs closely parallel with rinderpest, so, possibly, Rubarth's disease may run parallel with a disease which used to be very prevalent formerly in cattle in this country, though it is uncommon now, namely, malignant catarrh.

There is a similarity: there are sudden deaths in some cases, violent catarrh and so on, but it long remained unknown how the disease was transmitted. Work that has been done since points out that the animal which is the dangerous one in this respect is not the ox but the sheep, which carries the disease in an inapparent form and is capable of transmitting it. This is merely a speculation on my part, but I could not suppress in my mind the possibility that this disease we have under discussion here to-day may be similar to malignant catarrh in cattle in respect of causation and natural history.

The position so far as the ordinary veterinary surgeon is concerned is that we have a new disease in this country which first-class workers, like Mr. Parry, under an enlightened organisation like the Animal Health Trust, can most profitably take up for investigation. This class of research is not very likely to be taken up by a Government Department because they are not directly interested in the economic bearings of such a pursuit. The subject needs careful study, and careful consideration and discussion among virus workers themselves before anything is recommended to, or put in the hands of, practitioners to deal with the disease in practice. I think that is all I need say to stop the discussion taking trends and meandering in directions which at this early stage may merely lead them to just hopeless confusion.

The Chairman: In order to ensure that Dr. Edwards's opportune statement really matures, and to prevent any further expansion into malignant catarrh or suchlike, Mr. Parry would like to reply to some of the comments which have been made up to now so that we shall not forget the points to which he refers.

Mr. Parry: Professor Pugh raised first of all the question of kerato-conjunctivitis. I am afraid I am not in a position to compare it in detail with the form seen in leptospirosis. The syndrome we get in hepatitis is merely a clouding of the cornea. It is the type of reaction seen in the infectious kerato-conjunctivitides of cattle and sheep and also of man. Whether it is pathognomonic I do not know. All I can say at the moment is that it seems to us set apart from the other syndromes of kerato-conjunctivitis in the dog which have been reported.

The question of inclusion bodies was also raised. I should like to stress again that we think inclusion bodies are a very valuable diagnostic aid. This was brought home to me particularly last week. A dog had died of acute virus hepatitis in 50 hours after infection. Examination at autopsy and of liver smears produced evidence so equivocal that we wondered whether it was virus hepatitis until I found one or two inclusion bodies in the sections, and other lesions made it clear that the dog had died of a peracute form of the disease. I know American literature in particular stresses the inclusion body method as being an invaluable diagnostic aid. In our experience it needs to be used with caution. We think that tests of the liver or tissues from a dead dog for hepatitis antigen are very much better; in fact, we have always found a positive antigen test in these cases.

I had hoped to avoid the question of the relationship with distemper because we do not pretend to know the answer. We did start investigating the distemper-hard pad syndrome during our own epidemic of "hard pad" two years ago. The material was collected and was in process of being worked out and evaluated when hepatitis descended on us and since then we have been rather fully occupied.

It is all so very complex unless one can deal with one virus at a time and reproduce with it the total experimental disease; if we can do that, I think we may be able to help to unravel the problem in time.

I have deliberately not touched on technique or principles. All the relevant information will be published and it represents some 60 experimental animals. We are certain that hepatitis kills dogs without any assistance from anything else.

With regard to the receiving end or host dog, we agree that it is probably most important. The problem is to measure all the factors which might be involved. In our diet we use whale meat and a cereal mixture of the main home-grown cereals which we grind ourselves. We use cod-liver oil and a source of the vitamin B complex. This in the light of nutritional knowledge should be adequate on an analysis basis. On the question of diet, data on other virus infections and from infectious hepatitis in man provide very little evidence that nutritional factors are likely to be important. In the light of present knowledge it would be difficult to produce a nutritional situation which would affect markedly the susceptibility of the dog to the virus.

Dr. Edwards raised the matter of transmission. We think it is largely by contamination of food and food vessels. We have no experimental evidence, though there is a certain amount of American literature on the subject. We have evidence that lice will harbour antigen and that material from such lice will set up mild infection in dogs.

Miss O. Uvarov (Ewell, Surrey): I would like to add my apprecia-

tion of the contribution of this interim report. When it was first of all discussed in London at the "Central" one rather felt that this was a new disease we were about to investigate. Now it is strongly upon us and I think that the average practitioner is completely befogged. Ten or 15 years ago we had distemper. If we came across it early enough we could treat it and the animal either got better or died. Now when we begin to make a diagnosis we have to think whether it is this, that or the other. What it will be like after Congress I shudder to think!

I should like to ask Mr. Parry what laboratory animals he has used in an attempt to transmit this disease, and the numbers and types? If he could give us a little more statistical information I think it would help us.

Were there any secondary invaders in the enlarged tonsils or in any of the lymphatic glands? On page four of the paper mention is made of a nasty outbreak during cold November months. Is consideration given to the fact that climatic conditions might create a predisposition or otherwise? Mention was made of one case of cystitis, which seems to mean that every time we get a cystitis case we are going to think "Can it be that?" Had there been any cases of nephritis? If so, how many and of what type? Has Mr. Parry any information as to the geographical location of this disease? Have outbreaks indicated that it is endemic in one part and not in another? I noticed from the slides that Mr. Parry has not found leukopenia in his dogs, whereas in a lot of American literature they attach much importance to that finding. I was rather hoping that from a serological point of view we might have something concrete. Has Mr. Parry dealt with blood sedimentation in his tests, and, if so, could that be an indication?

I am afraid that Dr. Edwards did not help a bit. I think he rather showed his knowledge of virus is very great. His appreciation of the clinician's point of view, particularly Mr. Bateman's, was not as great as one might have thought. While realising that Rubarth did the original work, can Mr. Parry say if this is really such a new disease, or is it one of the co-existing, delightful viruses that sits for some time and, due to some form of stress or some unknown factor, comes up very conveniently so that we can have a paper about it at Congress?

Mr. Parry: Regarding laboratory animals, we are a little remiss on this score because we have not got a good small-animal house yet, but the virus will infect kittens, and chick embryos. We do not know whether it will in mice, and particularly suckling mice in which we are very interested, or in hamsters, and so on. We are organising this work at the moment but I am afraid I have no real data yet. In our experience, the disease does not occur in ferrets, although it is possible that ferrets may have an inapparent infection. I say that advisedly, because it is said that there is a hepatitis virus which will kill ferrets. We are going to see if these strains will kill our ferrets, but at the moment the matter is *sub judice*.

Regarding the tonsils and enlarged lymph nodes, one does not always get an enlargement of the tonsils, and when present, swabs may or may not be positive for streptococci; we have not recovered bacteria from body lymph nodes.

We have been watching the matter of climatic conditions. It is a very difficult thing, however, to put this on a strictly experimental basis so that one can control the environment and get really worthwhile data. It is something we should like to do because we think that stress such as exposure to cold might be a possible precipitating factor.

With regard to the case of cystitis, that was just an odd case sent to us for *post-mortem* diagnosis I think you can forget it in regard to clinical diagnosis in practice. I mentioned it merely to illustrate how mild hepatitis can occur as a "background" disease, if I may put it that way. We have no experience of nephritis but the Americans maintain that one of the chief effects of this virus is a chronic nephritis. How that ties up with what we have been talking about I do not know. We are at the moment in process of examining all our material on kidneys to see if it fits in with the American reports.

We have made no attempt to carry out any field survey, so we have very little evidence on geographical location. It is fairly widespread in East Anglia and the South-East of England. It has been reported from Glasgow, and of course the workers at Burroughs Wellcome have had much experience, probably more experience of the South-East of England.

Regarding blood changes, the North Americans lay great stress on the leucopenia and they would use it as a diagnostic criterion. We have not done a comprehensive series of blood examinations, but those we have done do not support what the Americans say. They seem rather apt to over-simplify these matters.

The sedimentation rate is markedly increased In a dog it is normally about 5 mm. per hour and it can go up to 40 mm., but that is not pathognomonic. The same sort of thing happens with hard pad. It probably merely indicates changes in the plasma proteins, and is a matter we are hoping to look into very soon.

The question of this being a new disease I will leave to those in the audience who have a much longer and wider experience than I have.

Mr. H. A. Crawshaw (Guildford, Surrey): I must confess that I have had very little experience of these conditions but I should like to congratulate Mr Parry on his paper because for me it has cleared up several points about which I have been concerned. I have really had only one kennel in which the condition has been diagnosed. I was particularly interested in the statement that a dog can be very severely affected with a nervous condition and recover, because I have destroyed two valuable dogs with what appeared to me to be quite hopeless nervous symptoms. I did persevere with one other dog which was an extremely valuable setter and it recovered quite shortly afterwards. What Mr. Parry has said does therefore appear to be the case, and a dog can recover from this condition although it seems apparently quite hopeless.

The two dogs which were destroyed were sent to a laboratory and were positive for Rubarth's; they had inclusion bodies in the liver, and the other *post-mortem* lesions associated with the disease. I therefore make a plea for any person who has a dog with what he considers to be Rubarth's disease to wait a little longer to see if the animal will actually recover. The two dogs I have mentioned might have recovered had I tried a little longer.

I should like to ask Mr. Parry if he has seen any evidence in young puppies of the conditions that I think some American workers describe. This is an oedema of the face and ears. I have seen it in one case which I considered to be Rubarth's disease, but it was unfortunately not confirmed in a laboratory. I should also like to know whether the keratitis is always bilateral. I have had one or two cases of dogs showing temperature reactions and they have been quite ill, but with a unilateral keratitis They have cleared up but the thought enters one's mind as to whether they were mild cases of virus hepatitis.

Finally, I should like to ask what materials are necessary for diagnosis when it is not possible to send the whole carcase? Are blood samples and pieces of liver required, and what other things might be necessary for this antigen test?

Mr. Parry: We have seen oedema of the extremities in our cases but never oedema of the head. We suspect that this virus affects vascular permeability. It is quite conceivable that there may be oedema of the head and neck and these signs, taken in conjunction with kerato-conjunctivitis, would, I think, be highly suggestive.

I am sorry I did not make it clear that the conjunctivitis we have seen can be unilateral.

With a dead animal the most certain method of diagnosis, but a tedious one, is the preparation of antigen from the liver. For that we require from 5 to 10 grammes—a small piece of liver put in a bottle without preservative. Perhaps I should add the rider that we are not equipped at the moment to do a vast number of diagnostic tests for liver antigen; it is quite a lengthy procedure. For histology, a small portion of liver ¼ inch thick in 10 per cent. formol saline is sufficient. For diagnosis in the living animal we require 5 ml. of clotted blood; if there is a positive antigen test well and good, but one can often be unlucky at the time of sampling. Examination of liver or kidney in the cadaver for antigen is a much more reliable method.

Dr. de Vries (Holland): Although I am not doing small-animal work I have had contact with a staff who are doing a little work on distemper diseases. They tell me that during two years they have dealt with a rather large number of animals suffering from distemper. They were inoculating ferrets, but in Amsterdam they could not find a dog that had died of this. That is so typical that I think there are many veterinary surgeons in our country who say that we have no distemper. I would ask you if in your country you get Laidlaw-Dunkin distemper regularly? If it is so, I can perhaps tell them in my country that they are making a mistake or that there is some difference in circumstances between Amsterdam and here.

Mr. Parry: It is not for me to say how frequent or infrequent Laidlaw-Dunkin distemper is. I am not in touch with it as closely as many practitioners, and I think Professor Pugh could answer your question much better than I can. But it is quite right to say that of the few dogs we have had submitted to us as distemper cases, a number have been found on investigation to have Rubarth's disease. With cases of dogs that are dying, which are suspected not to have typical Laidlaw-Dunkin distemper, one should look for Rubarth's disease. I will ask Professor Pugh to reply to the question whether Laidlaw-Dunkin's disease is still with us.

Professor L. P. Pugh: I do not retract anything from what I

said in my opening remarks. It is my opinion that at the present time in this country, if a dog is presented for *post-mortem* examination with the history of having died from distemper, it may well be either Laidlaw-Dunkin virus distemper, Rubarth's disease or so-called "hard pad" disease. I certainly agree that the typical Carré distemper with secondary bronchi-septicus infection has not appeared so frequently of late years, but I am equally certain that all the three diseases just mentioned have been mixed up clinically for many years in this country. It was not till Laidlaw and Dunkin sorted out one of them that we began to suspect that the distemper syndrome was composed of a number of diseases due to different viruses.

Mr. H. H. Skinner (Pirbright, Surrey): Anybody who has had experience of working with other virus diseases will realise the great difficulties under which Mr. Parry is working in having no suitable experimental animal. We must hope that he will soon be able to adapt the virus to one of the more commonly used laboratory animals and that he will then find his task very much easier.

In his paper he rather stressed the value of the test for antibodies based on the complement fixation reaction. He tells us that he cannot detect antibodies during the first fortnight, or even 10 weeks after infection in some animals, and that others, several months after infection, might lose the antibody content in their blood and yet still be immune to artificial infection.

There is one point I want to make in this connection, of which he is probably fully aware. The test is of course able to detect only a certain level of antibody, and I rather anticipate that when he has developed the use of other experimental animals he will find that the antibody can be detected very much earlier in the disease and that it will also remain for very much longer in the recovered animal.

I can see that at the moment one of his greatest difficulties is to determine whether animals which he uses for experiments have previously been infected with the virus, and until he has a very sensitive method for detecting antibody he might be seriously misled by results he obtains from his experimental dogs. I think we must hope that he will soon develop techniques which will be more sensitive than the present ones he has for detecting antibody, thereby ensuring that he has perfectly normal animals for his experiments.

Mr. Parry: I am sorry that Dr. Larin is not here, because the serological test is entirely his development. We are rather careful to explain that it is only serum antibody that we know anything about, and that we know nothing about tissue antibodies at the present time. The question concerning experimental animals is, of course, an extremely important one, and we are hoping to make developments as soon as possible. We have been fully occupied with the dog in the investigation to establish that the virus would produce the complete clinical disease.

I agree whole-heartedly with the point about knowing whether animals are susceptible or not. We have been lucky. Twelve months ago, all the young puppies we bred in our stud compound were susceptible as far as we could tell; they got the disease when inoculated. Now, on an average, only a quarter show clinical disease when inoculated. It is no use looking for serum antibody or blood antigen to explain this difference.

It is a very difficult matter to detect the non-susceptible animal before inoculation. As you are aware, it is a cardinal problem in many of these virus diseases.

Mr. P. S. Hastie (Ashford, Kent): My knowledge of this subject is very limited. To my knowledge we have seen only one possible case in Ashford—though that is probably not the dogs' fault but our lack of diagnostic ability!

I have been struck by several points while listening to Mr. Parry and the other speakers. I have always been under the impression that what determined the constant flow of a virus infection was the carrier animal, and that under different stresses and strains susceptible animals having come into contact with this disease, an infection could develop into a clinical case. Arising from that, I should like to ask Mr. Parry, with regard to the outbreak in his kennels, how long had the kennels been closed before the infectious hepatitis breakdown? In other words, was there any important interval from the time the last dog entered his kennels? In one particular instance he has mentioned, one dog suddenly became very ill and died very rapidly; all the other dogs, and presumably the one concerned, had given negative serological reactions.

The most important point, to my mind, was the suggestion that the spread of the disease might be associated with helminthiasis, and without carrying to absurdity the principle of fleas having smaller fleas on their backs *ad infinitum*, is it at all possible that the transmission of tapeworms by lice might determine that the tapeworm egg could carry a virus in its transport through life? *cf.* "blackhead" in turkeys.

I think I should mention here that the Animal Health Research Station, especially the Small Animal one, worked very hard to begin with under the most adverse conditions. The workers were erecting buildings themselves at the same time as they were investigating disease. Supplies and facilities were short but these conditions were no deterrent, and the results are all the more commendable. I thank Mr. Parry for his paper, which was most enjoyable.

Mr. Parry: In the original epidemic we had 38 dogs which had been on the premises for about six months. The premises had previously been a prisoner-of-war camp, and to our knowledge had never deliberately housed dogs. We started, knowing the risks without perimeter fencing. We could not keep stray animals away at night, and we had evidence when snow was on the ground that stray animals could come round the kennels, although there was no dog-to-dog contact. Where the epidemic came from, I do not know. Of the 38 dogs, four were greyhounds that Mr. Bateman had kindly let us have; 34 were bred on our premises and, as far as I know, had not had hepatitis although their dams had had hard pad.

With regard to the question of transmission, I do not know the answer. We have no evidence. Rubarth draws attention to the fact that many of his acute cases were heavily affected with roundworms. We are trying to keep the dogs free from all worms, but it is very difficult.

Mr. P. Turnbull (Midsomer Norton): I should be glad to have Mr. Parry's opinion on one or two points that have been raised. I am rather interested in this intermittent appearance of virus and antigen in the blood.

Last spring, in what one might almost call a "flying" kennel of greyhounds, a number of saplings and a perhaps rather less number of dogs from tracks developed, usually within a week of admission to the kennels, a febrile reaction with temperatures going up to 105° to 106° F.; and the symptoms involving pulse, heart and temperature were very similar to those of the syndrome mentioned earlier. No fatalities occurred at that time, although there have been one or two odd sudden deaths since. I could discover no satisfactory explanation for these deaths. We did examine the cadavers, but did not think of a possible connection with contagious hepatitis. The animals were in many cases housed in a range of kennels heavily infested with dog ticks (not lice). I was of the opinion that we were dealing with a specific infective agent which might well be carried through generations of ticks.

One symptom we observed, particularly in the first outbreak, and which has not been mentioned in connection with "Rubarth's disease," was a peculiar stiffness and extensor muscle rigidity which affected not merely the hind limbs, but the fore limbs as well. We were suspicious of *L. canicola*, but I think I can say definitely there was no other symptom or *post-mortem* lesion suggestive of this.

Other dogs have continued to be introduced. And more recently, so-called "hard pad disease" cropped up, as it often has before in these kennels. Fresh introductions into the kennels put up a similar febrile reaction within seven days of arrival, and many of these, in due course, usually within three or four weeks but sometimes nearly five weeks later, proceeded to develop a "hard pad" syndrome which appeared to be modified. Although I find that "hard pad" does seem to vary in different outbreaks, we have never had quite such significant changes in the symptoms as we had in these instances. Dogs appeared to become seriously ill much sooner after the initial febrile reaction. They seemed to go down with the more severe and unmistakable symptoms much earlier in the course of the disease. Within five or six weeks of the first malaise some of the survivors had put up pad lesions on no fewer than three occasions. If one were lucky these would subside, and within a week or 10 days they would be gone; and then one would go through the whole performance again, with a correspondingly weakened animal. In one case a bitch lived on for six weeks, during the last three of which the most one could say was that life was just maintained. The third time she put up hard pads, these remained "up" for 10 days. They seemed to make no progress. There was no tendency to shed or scale. A slow decline set in, and she died with the pads still extremely hard and without convulsions.

I should be very glad to know if Mr. Parry thinks it might be worth trying to find out whether dog ticks can harbour the hepatitis virus, possibly through successive moults, and whether, therefore, greater difficulties might be expected in infested kennels.

Secondly, if he thinks this curious change in severity, with repeated pad hardening, in "hard pad disease" could occur as a result of prior infection with Rubarth's virus; whether he con-

siders the two diseases together could present us with an even worse syndrome than that usually encountered in "hard pad" alone.

Mr. Parry: We have no evidence on tick transmission; we only have it on lice. I should hasten to add that with the lice it was an accident in that they came on two dogs which were sent to us, and I regret to say that the lice were not spotted during the isolation period. We have not deliberately tried to keep lice and we have no ticks. It also raises the question of the rôle of biting flies and mosquitoes, and we are hoping to have insect-proof kennels to raise our puppies in and conduct our infection experiments. In the light of what is known about so many of these parasites it is very important to check that.

I am unable to say anything about the muscle stiffness. If I could see the animals and examine them clinically I could get perhaps a clearer picture in my mind. We have had one or two cases, but they are very difficult ones. Are the spinal reflexes abnormal?

Mr. Turnbull: No, sir.

Mr. Parry: I do not think I can help about the recurring "hard pad." All I can say is that there can be recrudescence of clinical hepatitis, but how and why we do not know. The situation with the Laidlaw-Dunkin and Carré virus is just as problematical at the present time.

Mrs. M. L. Harris (Guildford, Surrey): I should like to congratulate Mr. Parry and Dr. Larin on a very interesting paper, which has presented a horrifying vista for the small-animal practitioner! I do not relish the thought of having to tell my clients that their dogs have recovered from a disease of which they are likely to remain carriers for 12 months. I do not relish having to tell them that their dogs are suffering from a disease about which I can really tell them nothing. The possibilities are altogether very unpleasant! It was different in the days when there was just distemper.

Mr. Parry showed us a picture of a dog suffering from motor disturbance, which he said recovered. I should like to know for how long there has been an apparent recovery of that dog, because dogs that recover from any kind of nervous disturbance have a nasty habit of breaking down a long time afterwards. I should also like to ask if there is a persistence of the cardiac symptoms. With hard pad one recognises a peculiarly bumpy heart in a dog, and it gives one an idea that a dog has had hard pad even if the owner does not know. I should like to know if there are any peculiar symptoms of the heart after Rubarth's disease which indicate that the dog may be a carrier of the disease?

Mr. Parry: I realise that we have posed many more questions from the practitioners' point of view than we have answered, but I would say that this position is by no means peculiar in the field of virus biology, and that we may have to revise our ideas concerning the natural history of many of these animal virus infections.

The photograph I showed was taken in early March, 1950. The dog showed the motor signs for 48 hours; on the third day it was sufficiently well to feed and get about. On the fourth day there was no motor deficit. Thereafter the dog was perfectly well until put down about a month ago, so it has survived nearly 18 months.

The cardiac signs do not persist once the general condition is improving. They are in fact a better barometer of the clinical state of the animal than the temperature. As the heart rate comes down and the pulse becomes of larger amplitude, slower and harder, so one knows that the dog is on its way to recovery even if the temperature is doing unusual things.

Mr. J. McIndoe (Dartford, Kent): I should like to ask Mr. Parry if he would care to elaborate on the symptoms, particularly bowel symptoms, shown by the non-fatal and milder type of Rubarth's disease; and also, if he has at any time in his experience associated Rubarth's disease with the acute haemorrhagic type of enteritis most commonly seen in greyhounds? The picture of the acute fulminating type, fatal in about 24 hours or even less, in my opinion completely fits that seen in many large greyhound kennels. I think it is time something was done about that type of case because for a long time now it has been very prevalent. In respect of what Mr. Bateman says, I think maybe in his well-organised kennels it is not very much in evidence, but in many others there is a considerable amount of it.

Mr. Parry: With the gastro-enteritis of virus hepatitis there is no blood in the stools in many cases; the stools may be soft. In the original epidemic they had a peculiar turpentine-like odour. Our stud kennelman told me 24 hours before any illness was apparent that we had distemper because he said he could smell it. He said he had observed it repeatedly in foxhound kennels. I think that a dog which gets sufficiently ill to void whole blood in the stool is dead before it voids the blood. I put it that way because if we get blood on the thermometer when taking a rectal temperature reading we look upon that, with other signs, as evidence that we have the infection. You occasionally get flecks of blood voided, but that is exceptional.

With regard to haemorrhagic gastro-enteritis of greyhounds and whether this is an expression of Rubarth's disease, we have not had an opportunity of investigating any epidemic of it, but there is a very strong *prima facie* case that it is in fact acute Rubarth's disease.

A Member: It has been suggested that the occurrence of the liver adhering to the diaphragm might be some evidence of previous infection with Rubarth's; and also that probably some of the fulminating cases in kennels, where serologically a number of dogs have been infected or show signs of infection, might be due to the breakdown of an immunity they have previously had. We have not been told, however, whether adherence of the liver to the diaphragm has ever been known in a fulminating case.

Mr. Parry: The adhesions between the lobes of the liver which we see in recovered cases of Rubarth's disease we do not see in an acute fulminating case, because I do not think there has been time. The interpretation we put on these findings is this: We know that in an acute fulminating case there is a fibrinous deposit over the surface of the liver. With a regression of the disease that deposit remains, becomes organised and actually fixes the lobes together, so that the adhesions occur only in animals that have lived for perhaps a fortnight. We have seen it only in animals that have survived for some months.

Recently, it has greatly strengthened our hand regarding the retrospective diagnosis of the experimental disease. We have had very mild or inapparent infections which on serological grounds appeared to be positive; at an autopsy several months later we have found these adhesions. As far as we know they have had no other disease in the intervening time, so we take it as retrospective evidence for the diagnosis.

A Member: Is there any evidence that the particular dogs that died of the fulminating case had had a previous infection?

Mr. Parry: Had the dogs that died of the acute fulminating disease in the outside kennels been previously affected under natural conditions?

"Yes."

Mr. Parry: I cannot answer that. We had not had the kennels under observation prior to the mortalities. We do not know for certain if these cases represent the recrudescence of a pre-existing infection, but it is a little difficult to explain their occurrence, in view of the subsequent evidence that all the other dogs in the kennels have had the infection recently, unless one assumes some such mechanism.

Mr. F. A. Crowhurst (Maidstone): I have been surprised that no discussion has centred round the suggestion that Rubarth's disease can cause sickness or death among suckling puppies. When Mr. Parry read a paper to the South-Eastern at Maidstone no mention was made of this fact. I should very much like to hear from Mr. Parry further on this. Even Mr. Bateman, with his usual scepticism of other scientific workers, has been moved to think there is something in this.

Mr. Parry: I rather deliberately did not raise the question of puppy mortalities because I thought I might get launched upon a rocky sea. All I can say is that we have definite proof, that is, pathological evidence with inclusion bodies plus serological data, that virus hepatitis can occur in newborn pups, dying in the period from two to four days after birth. We have actually had virus antigen in a puppy that never suckled and died within six hours of birth, but in this case there was no obvious pathology. I find the morbid anatomy of the newborn pups rather difficult; the pups are small, and perhaps we do not quite know what to look for. We have had four cases of acute hepatitis with peritoneal effusion in puppies dying in the period of from 48 to 96 hours.

The Chairman: I think we have had a very interesting discussion but we shall now have to bring it to a close. In doing so, there are one or two points I should like to make.

I am quite satisfied now that a former opinion of mine in regard to this matter was not as well founded as I thought. I was under the impression that with distemper, as in other virus diseases we might have met with, the number of variables of the virus or the so-called types might be proved by time to be different viruses.

This condition of hepatitis has brought back to my mind a number of cases where the *post-mortem* picture as described by Mr. Parry has been seen by myself over a fairly long period of time, but only in occasional cases, and in which we were unable to give any opinion as to the cause of it. Indeed in some of the cases where *post-mortem* findings were not quite so pronounced we felt that the animal had played a really dirty trick on us; we could find nothing to account for death and thought that the animal had had no right to die! I think that position may still be present in the minds of some practitioners, and it will at any

rate afford us an opportunity of giving some answer to the difficult problem or conundrum put to us by the question, "What did it die from?" We just cannot take refuge as can sometimes be done in the case of human beings when there has been a *post-mortem* and the coroner takes refuge in that old statement "Death from natural cases." We have to go one better!

There was one issue raised by Mr. Bateman that potential food factors might have something to do with the onset of this infection. Obviously they can have no direct exciting cause, but surely the normal resistance of individual animals as in man does depend to a very great extent on food factors, and I think we cannot dismiss that aspect completely as far as predisposing causes of the trouble are concerned.

With regard to the existence of Laidlaw distemper in this country, I may say that it exists very considerably in the London area at the present time but not to the extent it did formerly. I feel that the explanation of that is a very simple one, and is the more extensive use of vaccination against the trouble in puppies of an earlier age and the reinforcement of that immunity by mild reinfection during the course of time.

I think the time has now arrived for me, on your behalf, to express our great gratitude to Mr. Parry and Dr. Larin for the paper they have produced and for giving us something to think about. It will give me food for thought for quite a time. We must thank them also for the lantern display. I think some of the slides made the whole exposition very much more clear to all of us. (Applause.)

We are indebted to Professor Pugh for his very excellent opening of the discussion. We know his caution, and he has not betrayed us in that respect either.

I therefore, on your behalf, extend to the authors, the opener of the discussion and all who have taken part in it our very hearty thanks. (Applause.)

Mr. Parry: I should like first of all to thank the Chairman for his kind remarks. They are particularly appreciated coming from one who has had so long and deep an experience in this field. I should also like to thank you all for the way you have received this paper and for the way you have put questions, perhaps letting me down not too heavily! I should like to thank Professor Pugh for the very generous way in which he opened the discussion and the very kind comments he made about Dr. Larin's and my work.

I should just like to say that this work would have been quite impossible—I do not know of anywhere else in these islands where it could be done—unless Dr. Wooldridge had spent a great many years and much effort in providing us with the facilities and sinews of war with which to carry on. (Applause.)

SLAUGHTERING BILL FOR NORTHERN IRELAND

A Bill is being promoted by the Northern Ireland Parliament which seeks power for the Ministry of Agriculture to be the licensing authority for slaughterhouses. Under this Bill it is proposed that a licensed slaughterhouse shall comply with certain conditions, such as siting, throughput, availability of transport and the number of stock in a particular area. The details of the Bill are not yet available (states the *Meat Trades Journal* in its issue of November 22nd), but Mr. L. J. Seward, Chairman of the Slaughtering Committee, who reported the matter to the Council, said it seemed that if the Bill went through, slaughtering by private individuals would be at an end. Having regard to other schemes which had had their inception in Northern Ireland and which were later introduced into that country, such as the Pigs' Marketing Scheme, the Committee felt that the Bill should be thoroughly investigated.

Mr. J. O. Little, Northern Ireland representative, explained that the Minister of Agriculture in Northern Ireland was agent for the Ministry of Food and as such was the slaughtering authority and distribution authority. At the outbreak of war the number of slaughterhouses in use in Northern Ireland was 127 and this had been cut down to 17. In the opinion of his colleagues, if the Bill was passed the licensing would be regulated by the Ministry of Agriculture instead of the local authority and the private individual would have no chance whatever of using his slaughterhouse again. It was likely to be presented for a First Reading in a fortnight and they had asked for time to consider it before the Second Reading. "We feel," he said, "that it is prejudicing the whole future of the trade and we are very anxious about it."

It was decided to obtain a copy of the draft of the Bill, which will be fully investigated by the Parliamentary Committee.

THE VETERINARY RECORD

December 15, 1951

DOG PRACTICE

DOG practice in post-war Britain, contrary to many gloomy prognostications, has found ever-increasing calls upon its services. Despite difficulties and increased costs of feeding and the higher initial cost of a puppy, figures for dog licences have steadily increased and are now over three millions. Of these dogs, more than ever are pedigree animals. Owing possibly to this higher initial cash value of the puppy there is a greater tendency on the part of the dog owner to make a hobby as well as a pet-companion of his dog and to take a pride in its achievements on the showbench or in working and obedience trials. Even the pet owner with no intention of showing will dissertate proudly on his dog's points and pedigree.

Inevitably, most of these animals are kept under urban or suburban conditions; they may of necessity be placed in boarding kennels while the family has its annual holiday, some are taken to shows and most of them exercise in parks and small green spaces, or are taken round the shopping centres on leads. This gathering together of dogs itself creates one of the biggest problems of dog practice, that of controlling the spread of infectious and contagious disease, a problem to which the dog-owning community itself increasingly is becoming alive. There is a widespread awareness to-day of the need for veterinary service. This offers an opportunity to which the profession must not fail to rise.

To all this there is a background of grave world issues, rising costs, scarcity and expense of suitable premises, drugs and surgical instruments, which demands from the veterinary surgeon in canine practice considerable initiative and resource if he or she is to produce a service worthy of its opportunities and still remain solvent. It is before this background that the achievements, duties, hopes and problems of modern dog practice must be reviewed.

There are encouraging signs that the achievements have not been and will not be lacking. Scientifically, dog practice is in a state of change and advancement both medically and surgically. Medically, we have been blessed (or cursed) with a plethora of new chemo-therapeutics and antibiotics which it is our duty carefully to evaluate and to retain or discard according to their showing in the field, bearing in mind that no antibiotic can reasonably be expected to prove effective in the face of incomplete or inaccurate diagnosis, but also that all good clinicians are enlightened empirics and that a chance empirical observation may be the basis of valuable stimulus to research.

Medically, also, research has recently elucidated some of the problems that bewildered us pre-war and materially changed our approach to them. The subject of virology of the diseases of dogs is one in which our conceptions have recently undergone radical change. This change, no doubt, is due in part to rapid alterations in balance, virulence, and relative incidence of the viruses themselves and in part to our failure to recognise diseases which have long been with us even if only of sporadic incidence.

Many puzzling cases, previously put down by the clinician as "atypical" distemper, are now recognised as "hard pad" or as contagious hepatitis. Even more important as an augury for the future, however, is the fact that the clinician has had his complacency severely shaken, is now fully aware

that the last word has not yet been said and that clinical observation is acutely needed in alliance with research before the answers are found.

In seeking these, it may be added, research into canine problems, not only in virology but in other spheres, can justly claim to have helped elucidate questions bearing directly on human medicine, and such co-operation in the laboratory and in the field cannot fail to be of benefit to both professions and to the public generally.

General recognition of the leptospiral syndrome in dogs until recently has lagged behind the state of affairs pertaining among our European colleagues, although we may take pride in the fact that the impetus leading to its widespread recognition came largely from practice itself. In this disease the veterinary surgeon can by advice on the handling and hygiene of the case, help to prevent the possibility of its transmission to human members of the household.

Preventive medicine, particularly in those kennels with previous unfortunate experience of infectious disease, is steadily assuming a wider importance, and while we look to research to produce the methods of inoculation, much more could and should be done by way of education and advice in feeding hygiene and general canine management. No single person is in a more favourable position to " put this over " by precept and example than the veterinary surgeon engaged in dog practice whether on the case at home, or in the kennels or " vetting " at the dog show. Moreover, in the fortunately fairly few diseases communicable from dog to man in this country, frequently it is the veterinary surgeon who has the first opportunity of making a prompt diagnosis and advising the household on preventive measures.

Surgically, the advent of the antibiotics has opened up new fields, particularly in bone and joint surgery where operations previously hazardous because of the difficulty of attaining and maintaining complete asepsis in a non-co-operative subject, are now rapidly becoming standard routine.

The development of a satisfactory method of artificial respiration during operations involving opening the canine thorax has pointed to a further line of advance in chest surgery although at present, owing to the comparatively small number of indications for its use arising from any one practice, this seems likely to remain largely a hospital procedure.

There is, however, a generally increasing interest in the refinements of pre-narcosis and anaesthesia and in the value of endotracheal intubation which, while no startling new developments have recently taken place, make for safer and more accurate surgery by lessening the fears of anaesthetic fatalities and giving better relaxation.

Economic conditions and expense of equipment have increased the tendency for two or more practices to share some of the more costly items and frequently for their principals to co-operate as a surgical team for operations requiring more than one qualified surgeon. This friendly co-operation, not necessarily bound by any financial partnership, seems to be one step that can help modern dog practice to keep abreast of its surgical responsibilities in the face of present difficulties.

Those difficulties show no indication of early future abatement, yet there is every sign that the British public will not readily be parted from its dogs, and while the dog continues to flourish as the assistant, companion, guard and in the case of the blind, guide, of man, so long must dog practice remain an integral and vital part of the service which our profession has to offer.

CLINICAL COMMUNICATIONS

AN UNUSUAL CASE OF OESOPHAGEAL OBSTRUCTION IN THE DAIRY COW

A. T. MORGAN, M.R.C.V.S., D.V.S.M.,
Carmarthen
and
ERIC I. WILLIAMS, M.R.C.V.S.,
St. Clears, Carms.

Subject.—Six-year old Ayrshire cow; calved, November 18th, 1950.

History.—This animal, with other members of the herd, was fed whole potatoes just prior to the evening milking on November 21st, 1950. Shortly afterwards she became restless and was seen to be salivating profusely.

Examination.—The cow was observed to be in a state of acute distress. There was slight ruminal tympany, and defaecation and urination were frequent, as was the regurgitation of large quantities of saliva.

Diagnosis.—Since palpation of the throat and neck region had failed to reveal any signs of obstruction, it was concluded that a potato had lodged in the lower third of the oesophagus.

Procedure.—The animal was suitably restrained and a probang, inserted via the mouth, was passed carefully, and without difficulty, along the oesophagus up to a point estimated to be about 6 to 9 inches short of the ruminal opening. Despite constant *steady* pressure on the probang, it was unable to move the obstruction.

The intramuscular injection of 2 c.c. carbamyl choline (1 to 1,000) having proved of no avail, it was decided, in view of the location of the obstruction, to perform a rumenotomy. The site was prepared in the usual way, and the skin muscles, peritoneum and rumen were incised. Insertion of the arm into the rumen revealed the presence of an afterbirth. This was followed carefully and part of it was found to be extending into the oesophagus. By steady traction this section of the afterbirth was withdrawn into the rumen—followed by the offending potato! After removal of both placenta and potato, the operation was completed in the usual way. The animal progressed favourably, complete recovery being reached in about 10 days.

Remarks.—The owner revealed that the cow had calved out in a field three days previously, and stated that he had seen no sign of the placenta. It is concluded that the animal had eaten the placenta which had not caused any apparent ill effects until the potato became obstructed in the oesophagus, and that attempts by the cow to expel the potato caused the regurgitation into the oesophagus of part of the placenta, thus aggravating the obstruction.

It might also be added that the writers are not " probang fans." Its use is only recommended when oesophageal obstruction occurs in the lower third and then with extreme caution.

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ANTRYCIDE METHYL-SULPHATE IN THE TREATMENT OF *T. CONGOLENSE* INFECTION IN DOGS

J. C. ALLEY, M.R.C.V.S.,
Zanzibar

Over the past year, five pariah dogs were brought in suffering from *T. congolense* infection and were given 0·5 c.c. of a 10 per cent. solution per 15 lb. bodyweight of antrycide methyl sulphate. All made excellent recoveries. No toxic

symptoms were observed and in each instance blood smears, taken 48 hours after injection, and onwards, were negative.

Two of the cases treated are worth special mention. In both, the dogs were emaciated and unable to stand—one was suffering from profuse dysentery—and it was considered that any treatment would be of little use.

Another dog, a fox-terrier type, suffering from *T. vivax* infection, was given, during my absence, 1 c.c. of a 10 per cent. antrycide solution, which was, I think, rather too high a dose. The dog succumbed an hour after being treated, showing toxic symptoms.

ABSTRACTS

Phosphorus Deficiency in the Grazing Cow*

In many parts of Ireland a chronic constitutional disturbance of grazing cattle has been recognised for some generations, and known locally by such names as "bog crook," "bocynamonia," "luanac" and "cropac." Its incidence is particularly high on the carboniferous limestone plain that extends over the midland counties. The condition is associated with pica, unthriftiness, stiffness, lameness—occasionally with osseous enlargement and in advanced cases with progressive anorexia and emaciation.

The first symptoms of "bog lame," to give it its most commonly used colloquial designation, usually appear during May, about two months after the cows have been turned out to pasture. Initially, only one or two animals are affected, and although there may be other cases later, it is rare for many members of the herd to be affected simultaneously. Symptoms invariably disappear during the winter some time after concentrate feeding has been commenced, but may reappear in the following grazing season after the cows have been turned out for a month or more. Milk yields on "bog lame" farms are low, and in some of the cows and heifers there is usually a history of anoestrus or of oestrus with repeated failure to conceive. Store cattle make poor growth and often appear stunted.

Of recent years it has been shown that "bog lame" is associated with phosphorus (P) deficiency. The present paper records a detailed study which had reference to (1) the calcium (Ca) and P contents of the vegetation on "bog lame" and on apparently normal farms; (2) the effect of supplying phosphates to cows grazing on herbage low in P, or of transferring the cows to pastures of normal P content, and (3) the value of blood analysis in detecting latent aphosphorosis.

It was ascertained that cows which had grazed P low herbage for about two months developed a low blood inorganic P level whether or not they exhibited clinical symptoms. Blood inorganic P values of the order of 2·0 mg. per 100 ml. were associated with pasture P contents of $< 0{\cdot}15$ per cent. P (dry matter). The serum Ca levels did not vary between animals on "bog lame" and normal grazings, and in both cases the dry matter of the pastures contained from 0·38 to 0·92 per cent. Ca. In certain farms on which the herbage P was $< 0{\cdot}15$ per cent. the width of the Ca/P ratio in the dry vegetation appeared to be proportional to the incidence and severity of symptoms in the cows. The ratio on "bog lame" farms ranges from 2·8 to 8·7/1, whereas on normal farms it ranges from 1·8 to 2·6/1.

When the cows were changed from "bog lame" to normal pastures, or when they were provided with a dietary supplement in the form of 2½ oz. of dicalcium phosphate daily, their blood inorganic P level was restored to normal in 7 to 44 days. In animals clinically affected the remission of symptoms occurred but was less rapid, and there were therefore animals still with symptoms but with a normal blood inorganic P. The provision of dietary phosphate was followed by the recurrence of oestrus in nine hypophosphataemic cows.

Blood inorganic P analyses are held to be of diagnostic value in aphosphorosis only if the animals have been on affected pastures for some time and have not received supplementary feeding. In such circumstances, however, the easiest method of detecting P deficiency in pasture is stated to be by carrying out inorganic P estimations on the blood of several of the grazing cows.

A. N. W.

* Observations on Phosphorus Deficiency in the Grazing Cow. O'Moore, L. B. (1950.) *Irish vet. J.* **4.** 198-209 and 218-227.

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Lead as a Nutritional Hazard to Farm Livestock*

A summary of data recorded at Weybridge indicates that approximately 0·2 to 0·4 g. per kg. bodyweight of lead ingested on any one day, either as the acetate, the basic carbonate or the oxide, is sufficient to cause death in calves up to four months of age. For older calves and mature sheep, however, the necessary dosage appeared to be of the order of 0·6 g. per kg. upwards.

In lead poisoning of sheep the pattern of symptoms is as follows. A slight transient rise in body temperature of up to 1·5° F. is noted the day after ingestion of the lead compound, followed later by progressive anorexia and reduced water consumption, persisting until death or disappearing with slow recovery. At this stage loss of condition is very marked, the sheep becomes dull and lethargic, and weak as judged by ataxia of the hind quarters on prolonged standing. No tetany is observed, but there is often evidence of abdominal pain.

In young calves, in many cases, acute symptoms differ markedly from those in sheep. In general, however, dullness and inappetence occur as well as abdominal pain with constipation, sometimes followed by diarrhoea. Usually, these symptoms do not arise until two to three days after a fatal dose, and in some cases the first symptoms to be observed are sudden bellowing and staggering, with rolling eyes and frothing mouth. In this phase, which can last up to two hours prior to sudden collapse, the calf is greatly excited, running at the walls of the stall and trying to climb them. Muscular spasms and tetany occur with increasing frequency until death. The phase of extreme excitement may, however, be short and subsequent collapse rapid, so that the calf is merely "found dead."

On *post-mortem* examination all sheep have shown extensive inflammation of the gut and damage to the kidneys. Similar changes are not constant in calves, in which inflammation of the gut is not always observed, and varies greatly in intensity, while in many cases kidney damage is only slight.

During the six-months period ended June, 1948, some 83 suspected field cases of lead poisoning in cattle were investigated by Weybridge, and 40 of these, involving 67 animals, were deemed to be positive according to the tissue and concentration of lead. Since only 25 veterinary surgeons appealed for laboratory assistance, it seemed apparent that the actual losses of cattle from lead poisoning throughout the country must reach a very high total.

* Lead as a Nutritional Hazard to Farm Livestock. V. The Toxicity of Lead to Cattle and Sheep and an Evaluation of the Lead Hazard. Allcroft, Ruth, & Blaxter, K. L. (1950.) *J. comp. Path.* **60.** 209-218.

These 40 positive cases, and a like number of previous cases, are analysed. They involved altogether approximately 113 young calves and 71 heifers and cows. In young calves the main source of lead incriminated was painted woodwork, death being due to ingestion by chewing or to licking off the flaking paint. Other sources included painted feeding troughs and utensils, tins of fresh paint, lead storage batteries, painted canvass or tarpaulins, and painter's materials left on rubbish dumps, including putty and red lead.

From long-term experiments and from pasture-level studies it is concluded that cumulative lead poisoning rarely, if ever, occurs in ruminants, which therefore in this respect differ from human beings. Although the average annual ingestion of lead by sheep on the plumbiferous area of Derbyshire must be high, there is probably no danger of any form of plumbism as a result.

It is emphasised that an unequivocal verdict of acute lead poisoning from analytical data above, without collateral evidence, is often difficult.

A. N. W.

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Ranunculin *

The vesicant in the Ranunculaceae is a water-soluble, steam volatile oil, protoanemonin, which is freed enzymatically from a glucosidic precursor when the plant is crushed. The glucoside has been isolated as crystals, the name proposed being " ranunculin." It is stable and a convenient source of protoanemonin. The glucoside appears to be present only in Ranunculaceae.

F. A.

* **Ranunculin: The Precursor of the Vesicant Substance of the Buttercup.** Hill, R., & Heyningen, Ruth van. (1951.) *Biochem. J.* **49.** 332.

REPORT

Nigeria: Annual Report of the Veterinary Department for the Year 1949-50

This report covers the period January 1st, 1949, to March 31st, 1950, and consists of an introductory survey by the Director, Mr. W. G. Beaton; reports by the Assistant Directors, Messrs. Morton, Littlewood and McCulloch; a report on the Vom Veterinary School by the Principal, Mr. G. Williamson, and an important section (unfortunately curtailed for publication) by the Assistant Director, Laboratory Services, Mr. R. W. M. Mettam. There is also a small section on the activities of the Livestock Investigation Centre by Mr. W. E. Hill, Senior Livestock Superintendent.

The Director stresses the extreme shortage of veterinary officers. Not only has the department not been able to recruit the additional staff sanctioned for the increased activities, but since 1948 there has been an actual loss of six veterinary officers and there are now no fewer than 14 vacancies in the sanctioned establishment. Senior men are retiring at the early age of 45, when they could and should be making a valuable contribution to the framing and execution of departmental policy. The matter is serious and the cause should be investigated. On the other hand, it is pleasing to note that the staff of the laboratory division is up to full strength, although one research officer has proceeded on leave prior to retirement. It has to be recorded with deep regret, however, that since the report was written Mr. Mettam has died, leaving another and difficult post to fill.

The main work of the department has continued to be the control of cattle epizootics by large-scale prophylactic vaccination, a policy which, since its inception a good many years ago, has paid handsome dividends. No serious outbreak of any of the major epizootics has been reported and only isolated cases have occurred. The total number of cattle given vaccination in all regions reached the surprisingly high figure of over four million. Of this number some 1,225,000 cattle were immunised against rinderpest in the Northern Region alone, using caprinised virus. Other inoculations in this region include 97,837 against contagious bovine pleuro-pneumonia, 379,680 against blackquarter, 14,839 against haemorrhagic septicaemia and 657,808 against anthrax. Vaccinations numbering several thousands were also carried out against the prevailing poultry epizootics: fowl typhoid, fowl cholera and fowl pox. In addition to this work of vaccination, many thousands of animals received treatment for helminthiasis, trypanosomiasis and epizootic lymphangitis. The castration of some 9,000 cattle, sheep and goats was also completed as a means of improving native breeds by the policy of selection.

There is no space in this review to go into further details of these field inoculations but their total volume reflects the greatest credit, firstly on the laboratory staff for manufacturing the many biological products in such large quantities and secondly on the field staff for their work, arduous and often monotonous, in the administration of these products.

The development of the industries in the various animal products, notably hides and skins, has continued to receive close attention by officers of the department and satisfactory progress has been maintained. Nigerian hides and skins are in large demand by oversea manufacturers for their high quality.

The full expansion of the livestock industry in Nigeria has been seriously hindered in the past through the lack of any Government policy on the question of whether the Agricultural or the Veterinary Department should be responsible for this work. A conference of agricultural and veterinary officers was held at the instigation of the Government to decide on the degree of responsibility to be assumed by each department for framing and implementing the policy of livestock development. This conference has proposed that in this development work the veterinary department should be responsible for " disease control and investigation, the movement of stock and its examination prior to slaughter, meat and animal products, control of artificial insemination, and the promotion of animal health in general."

Unless the last line, " the promotion of animal health in general," is a loose way of saying the promotion of animal productivity in general, the situation in Nigeria is no different from what it has always been, with the development of the industry in the hands of two separate departments. The sole purpose of any scheme of livestock development is surely to increase to the fullest possible extent the productivity and economic value of stock. In the attainment of this object there are no items of greater importance than proper breeding, feeding and management. But these important duties have been delegated to another department. Without a sound and fully integrated policy of development devised and managed by a single authority there can be no real and sustained progress of the livestock industry, which is of great and vital importance to the social and economic well-being of the native populace and, indeed, to the country generally.

Research.—Only an abridged report of the work of the

Laboratory Services is published. This is a very great pity. Veterinary research in tropical Africa is a matter at once of inexhaustible depth and perplexity and of supreme economic importance, and a free and full exchange of information between research workers in all colonies is a matter of supreme moment. Often the only vehicle for this exchange is the annual reports of the various laboratories and any abridgment of essential details of research work should be avoided if at all possible. It is unfortunately necessary, because of lack of staff, for a great deal of the time of research workers in the colonies to be spent in the production of the large quantities of the various vaccines which are needed by field organisations, and in this regard Vom laboratory is no exception. During the period of 15 months covered by this report no fewer than four and three-quarter million doses of vaccines were made and issued.

Some research work was done in an effort to make caprinised virus vaccine more suitable for calves and yearlings of the Zebu breed. Trials with lapinised virus were also conducted.

In the use of lapinised virus, however, it was found that when the virus was inoculated direct into cattle the immunity resulting was not strong enough to withstand a test dose of bovine virus. Better results were obtained when lapinised virus was first passed through goats before inoculation into cattle. A stronger immunity was thus obtained.

Among the non-Zebu breeds of cattle which frequent the tsetse fly belts of Southern Nigeria and which possess an unusually high degree of susceptibility to rinderpest, it was found that the results with lapinised virus in all but the dwarf Muturu breed of cattle were very satisfactory. In Muturu cattle, however, the reactions were much too severe and further attenuation of the virus will be necessary before it can be used with safety among these abnormally susceptible animals.

Research into trypanosomiasis was confined to testing the value of antrycide methyl sulphate as a curative agent in cattle and pigs. It was found that the drug in single doses of 5 mg. per kg. cured 10 out of 10 cattle infected with *T. congolense* but the same dosage gave less satisfactory results against *T. vivax* infection. In this case 9 out of 12 animals relapsed and even a second dose did not cure all, three relapsing once more.

An interesting point proved by Mettam was that strains of trypanosomes rendered resistant to antrycide could still be successfully dealt with by dimidium bromide. Two outbreaks among pigs were dealt with and injections of antrycide enabled the pigs to be reared successfully to bacon weight.

In the biochemical section of the laboratory, work was undertaken to find out the degree of mineral deficiency of the soils of Nigeria. So far, it has been determined that soils in the neighbourhood of Vom are not lacking in calcium, magnesium, phosphorus, copper, cobalt, iron, manganese and molybdenum.

In the Cameroons Province of Southern Nigeria, Mr. McCulloch, Acting Assistant Director, records cases of staggers among cattle which quickly responded to subcutaneous injections of magnesium sulphate. He also records the occurrence of licking disease among calves at Buea, which responded well to traces of copper sulphate.

Clinical Work.—At the many animal clinics which are now established throughout the country a vast amount of work was done in treating many thousands of animals for wounds, lameness and general ailments. Helminthiasis and trypanosomiasis are being treated on a large scale. In the Northern Region alone 170,000 animals received phenothiazine wireworm mixture and 34,000 cattle were treated for trypanosomiasis.

Veterinary Education.—Shortage of teaching staff seriously curtailed the activities of the Vom Veterinary School.

It has now been decided that activities at the Vom school are to be confined to the teaching of African assistants only and the training of assistant veterinary officers is to be carried out at the University College, Ibadan.

In Nigeria, with its large nomadic cattle population and its grazing grounds, often inaccessible to motor transport, it is important for the conduct of the large-scale scheme of vaccination and for other work that there should be an adequate staff of African assistants, and if the Vom school can provide the training for these men now wanted in large numbers it will have fully justified itself.

Livestock Improvement.—Some additional evidence has accumulated that the N'dama breed of cattle—a breed originating from French Guinea—possesses an appreciable degree of resistance to trypanosomiasis, which is heritable. This has proved to be the case at any rate when these cattle are exposed in *G. palpalis* country. The resistance of this breed when exposed in *G. morsitans* country has not yet been tested. N'dama cattle imported from French territory are being used by the Agricultural Department to improve the indigenous breed of dwarf cattle which inhabit the tsetse belts of Southern Nigeria.

This is but a short summary of the work done by the Veterinary Department during the period under review. Much valuable work has been done and the Director and staff of the department are to be congratulated on maintaining essential field work at such a high level in spite of the serious shortage of professional staff.

W. W. H.

SIXTH INTERNATIONAL CONGRESS OF COMPARATIVE PATHOLOGY

The sixth International Congress of Comparative Pathology will be held in Madrid, Spain, from May 4th to 11th, under the Presidency of Professor Enriquez de Salamanca. The Congress is concerned with the study of diseases common to man, animals and plants.

Papers will be presented to the following sections:—

1. Mycosis in comparative pathology; 2. Trace elements; 3. Factors influencing senescence; 4. Insecticides, and 5. Bacterial symbiosis.

In addition, there will be a symposium on the aetiology and pathology of arteriosclerosis organised by the Council for the Co-ordination of International Congresses in the Medical Sciences. Full particulars can be obtained from the Secretary to the Congress, Professor Carda-Aparici, Calle Fernando VI. 8. Madrid, or from the Hon. Secretary, British National Committee, Professor R. E. Glover, Department of Veterinary Pathology, 42/44, Bedford Street North, Liverpool, 7.

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German railway authorities have just made it clear to a firm in Goettingen which prepares anatomical specimens that as far as they are concerned a horse is a horse. The firm exported to Luxembourg the skeleton of a large and imposing charger, carefully wired together and mounted on a stand. The skeleton was required for veterinary instruction. The transport bill contained an item of 52 francs for "wagon-cleansing charges." The firm objected, reinforcing their remarks with a number of physiological observations. The railway replied that it was not concerned with physiology but only with the clearly stated terms on which horses were carried by rail. It got the money.—*Daily Telegraph.*

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Weekly Wisdom

No man should be free to take from the land more than he can profitably employ in the enrichment of it and in his own support, in adequacy and comfort.—Report of Royal Commission into the pastoral industry of Queensland.

REVIEW

[**Meat Hygiene.** By A. R. MILLER, D.V.M., LL.B. Henry Kimpton, 25, Bloomsbury Way, London, W.C.1. Pp. 312 + 108. 52s. 6d.]

This text-book has been prepared as a guide to the study of meat inspection in the U.S.A., where the author is the chief of the Federal Meat Inspection Service, and will no doubt prove of use to veterinarians and veterinary students in that country. Elsewhere, its appeal will be limited to those interested in the wider aspects of meat inspection, for its value as a work of reference; mainly because of the 90 pages which form the appendix and which contain the Food Inspection Acts and the Federal Meat Inspection Regulations.

A chapter of five pages is devoted to *ante-mortem* inspection, and one of 55 pages to *post-mortem* examination. The latter, however, contains 22 pages on slaughtering and dressing of carcases and only 33 pages on diseases and other conditions which are likely to be encountered by the meat inspector, with a guide to the judgment of meat as to fitness or otherwise.

Tuberculosis is dealt with in some 60 lines of script and two illustrations (depicting tuberculous bovine peritonitis and pig's spleen). The progress of the campaign for the eradication of tuberculosis in cattle in the States is reflected in the fact that whereas, in 1920, of the cattle inspected 2·05 per cent. were retained for detailed inspection on account of this disease, this figure was reduced to 0·18 per cent. in 1948, whilst condemnations were 0·386 and 0·010 per cent. respectively. The relative insignificance of the problem in America might be deemed to be reflected in the space allocated to it in the book were it not for the fact that most other conditions are dismissed in less space; actinomycosis has merely 20 lines and pericarditis only six. Mange, however, occupies over a whole page (55 lines), lung worms nearly as much and "ox warbles" are given 26 lines.

Under methods of slaughter, humane slaughtering is not discussed but the captive bolt pistol is mentioned by reference to the fact that the bolt may carry particles of skin and hair into the brain and this contamination will render the brain unfit for food. As the Productivity Team from the British Meat Packaging and Processing Trade reported after its visit to the U.S.A., "Humane slaughtering methods seem to be comparatively unknown in the U.S.A., at least that is the impression one gets." It is remarkable that, in so enlightened a country, the welfare of animals appears to have received so little consideration.

Trichinosis is widespread in America, and a whole chapter of 24 pages is given up to this disease and the life history of the causal organism, which is well illustrated by large diagrams. This chapter, however, appears to be an attempt to justify the Federal Meat Inspection Service's repudiation of all responsibility for ensuring protection of the public from the risk of infested fresh pork. The onus of safeguarding itself against disease by cooking all raw pork purchased, by raising the meat throughout to a temperature of 137° F., is placed on the general public. This would hardly be deemed a sound and practical measure by public health workers in this country, and although it may be considered, perhaps, to dispose of the problem in theory, it is very doubtful if, in practice, it achieves this object. Prepared pork, which is normally eaten as purchased, is required to be subjected, during preparation by the trade, to prescribed methods of refrigeration, heating or curing designed to kill the parasite in accordance with the Federal Meat Inspection Regulations detailed in the appendix at the end of the book.

A chapter on "Physical and Chemical Characteristics of Meat and the Principal Organs" contains little which is likely to be unknown by the British veterinary surgeon or student. Although the book is said to be written mainly for the veterinarian and the veterinary student, this chapter is chiefly a recital of anatomical, histological and physiological details with which all students in this country would be familiar before proceeding to the study of pathology and subsequently to that of meat inspection. The inclusion of such a chapter is all the more notable since there is no serious attempt to deal systematically with the all-important subject of pathology. If it is considered necessary to deal with the histological features of tissues, this should surely have been followed by a serious attempt to treat of the all-important subject of the lesions of disease.

Much space is allocated to descriptions and illustrations of materials, such as spices, which are incorporated into meat products—almost as much, indeed, as that given to the consideration of disease.

"Sanitation in Plant Operations" is extensively discussed, and detailed descriptions are given of many insect and rodent pests and measures for their control. The illustrations of various insect pests are undoubtedly, from the point of view of instruction, the most useful of any in the book.

Whilst the work contains much of ancillary interest to the British meat inspector, it can hardly be recommended as a text-book for students of the subject of meat inspection in this country. The high level of bacterial control exercised in the American food industry is well appreciated here, and those sections which deal more specifically with "clean food production," in its more limited sense as applied to the meat industry, will probably be read with greater interest by British readers than most of the other aspects of "meat inspection" which the author has set out to expound.

BRISTOL UNIVERSITY'S FIVE-YEAR PLAN

A five-year plan, beginning next year, for the further development of Bristol University was announced on November 30th, when the University Council presented its annual report to the Court.

It was stated that development would be limited in the immediate future to the construction of the engineering school and to the development of the veterinary school. Provision was being made for extensive academic developments at a later stage.

* * * * *

EXPERIMENTS ON LIVING ANIMALS

A return of experiments performed on living animals during 1950, published on December 5th (Stationery Office, 4d.), shows that a total of 1,779,215 experiments was performed by 2,774 persons holding licences. Just over half of 1 per cent. of the experiments involved the use of cats, dogs, horses, asses and mules. More than half of these were performed under anaesthesia throughout, and many of the operations were of a minor character.

Of 2,689 experiments performed upon dogs without anaesthetics, over 60 per cent. involved the testing and standardisation of substances for use in treating canine diseases. The majority of 78,192 experiments relating to cancer investigations were performed upon mice. More than 1,000,000 experiments involved preparing and testing substances intended for use in treating disease in man and animals.

* * * * *

BRITISH SHOW JUMPING ASSOCIATION

At a meeting of the above Association held in London on December 5th, Lieut.-Colonel C. T. Walwyn was re-elected President and Lieut.-Colonel M. P. Ansell Chairman. Mr. J. Taylor was elected a Vice-President.

A satisfactory financial position was announced. The membership is now just on 5 000 and more than 6,000 horses are registered. Of these registrations 500 are in grade A.

The 88th Convention of the American Veterinary Medical Association

I was privileged recently to attend the 88th Annual Meeting of the American Veterinary Medical Association and to make a small contribution to its proceedings. The following notes may be of interest to N.V.M.A. members.

The meeting was held in Milwaukee, Wisconsin, from August 20th to 23rd and attracted a record attendance of no fewer than 3,200 persons of whom some 1,700 were veterinarians. Despite the difficulties of dealing with so many people the proceedings were conducted with great precision and smoothness and the outstanding success of the Convention is a tribute to the energy and resourcefulness of Dr. Hardenbergh and the other officers of the A.V.M.A.

The opening session of the Convention took place at 9 a.m. on Monday, August 20th, and between then and the closing session at 12.30 p.m. on Thursday, August 23rd, no fewer than 70 papers were presented, followed immediately after the conclusion of the main meeting by another nine on Military Requirements and Civil Defence Planning. In addition, six films were shown and in five closed circuit television sessions, numerous very successful demonstrations dealing with anaesthesia, surgery, *post-mortem* technique, etc., were presented.

In opening the Convention, Dr. Coffee, President of the A.V.M.A., stated that an endeavour had been made to arrange a programme which would in effect constitute a refresher course for veterinarians of all interests. And so it proved to be. Throughout the meeting the main emphasis was placed on the scientific sessions which proceeded continuously from 9 a.m. until 5.30 p.m. or later and which were followed each evening by meetings of constituent societies, alumnus associations, etc. Less importance was attached to the social side, except for the ladies, for whom a full programme of activities had, of course, been arranged. The general social events were limited to a short Gemütlichkeit or "get-together" from about 9 p.m. to 11 p.m. on Monday evening, a visit to the Wisconsin State Fair from 8 p.m. to 11 p.m. on Tuesday and the President's reception and dance from 9 p.m. to 1 a.m. on Wednesday.

To the interested observer such as myself, a noticeable feature of the Convention was the very high proportion of younger veterinarians attending it, many of whom claimed that they relied upon it very largely to keep them abreast of recent developments and who openly asserted that for this reason alone they always attended.

For some years the A.V.M.A. has adopted the system of conducting two sessions of papers simultaneously at its Convention so that members have a choice throughout the meeting. This system was again used at Milwaukee and operated very successfully. On Monday and on Tuesday morning there were simultaneous sessions dealing with General Practice and with Research; on Tuesday afternoon and Wednesday morning the sessions were on Small Animals and on Poultry; and on Wednesday afternoon and Thursday morning, on Surgery and Obstetrics and Public Health. The films were projected separately during Monday afternoon and were repeated during Thursday morning. Thus it was ensured that at any time there was always something of specific interest to the individual member no matter how specialised his work.

As an additional item of interest an impressive educational exhibition was arranged concurrently with the Convention. This featured demonstrations dealing with Brucellosis, Histoplasmosis, Leptospirosis, Mastitis Control, Meat Inspection, Milk and Food Sanitation, Veterinary Public Health, Bacterial and Parasitic Diseases of Poultry, Avian Infectious Bronchitis, Avian Pneumoencephalitis, Graduate Training and Research, Radiologic Aspects of Anatomical Abnormalities, and A.V.M.A. publicity. All of these exhibits were very well prepared and presented and created considerable interest. There was also, of course, a very comprehensive commercial exhibition supported by some 60 commercial houses with displays extending over two large halls. This, too, was well arranged and attended.

Scientific Papers

The scientific papers ranged widely over the whole veterinary field and it is not possible here to do more than touch on a few of them. In the general practice session papers dealt with bovine acetonaemia, vesicular stomatitis, antibiotic feeding, swine fever vaccination, swine erysipelas, light horse practice, and biological warfare and civil defence. In addition there was a symposium on all aspects of brucellosis.

In dealing with bovine acetonaemia, Professor Moore, of the Michigan Veterinary School, described a practical method of continuous dextrose intravenous drip medication which is giving excellent results and which is claimed to reduce the number of relapses to a minimum. Mention was also made in this session of spectacular results being obtained by Shaw in Maryland with cortisone in this condition. Vitamin medication was agreed to be of no value.

Dr. Catron, Professor of Swine Husbandry at Ames, Iowa, and one of the foremost exponents in this field, summarised the antibiotic feeding situation very clearly and concisely. Pigs, poultry and, to some extent, calves have all shown marked response to such feeding. Dr. Catron regards antibiotic feeding as the greatest advance in pig nutrition in recent years and produced impressive figures of responses especially in runt pigs.

In immunisation against swine fever, the availability of new vaccines which have been produced by passage through rabbits was announced at the Convention. With the help of these vaccines it is hoped that eradication of this disease, now apparently endemic in the country, will become possible.

The section on research featured papers on experimental pyelonephritis in cattle, rabbits and mice, experimental production of hyperkeratosis (X-disease), induction of swine erysipelas by skin scarification, effects of oestrogens on bitches, Newcastle disease virus and poliomyelitis virus in monkeys, vesicular stomatitis in sheep, duration of immunity with S.19 vaccine in brucellosis, *Brucella abortus* infection in pigs, brucellosis surveys, virus anaemia (ictero-anaemia) of pigs, swine influenza, Beta haemolytic streptococci in pigs, digestive disturbances in pigs, thyroxine and glucose relationship to the motility and conception rate of bovine semen, "Rh" and iso-immunisations in thoroughbred horses, liver fluke control, lungworm in sheep, and the use of antibiotics in fowl cholera.

An interesting observation by Dr. Shuman, of Washington D.C., indicated that swine erysipelas can be transmitted consistently from pig to pig by skin scarification. Inability to transmit the disease with any regularity has often held up research on this disease in the past.

As in Britain, concern is felt in America at the lack of definite knowledge of the duration of immunity induced with strain 19 vaccine and considerable interest was aroused, therefore, by Drs. Manthei, Mingle and Carter, of the U.S. Department of Agriculture Experimental Station at Beltsville, Maryland, who announced that comprehensive work on this aspect of the vaccine was in hand. Dr. Mingle will be remembered in Britain as the U.S. veterinarian who flew the Atlantic in the early years of the war to give us manufacturing details of S.19 vaccine.

In liver fluke studies, Drs. Batte and Swanson, of Gainesville, Florida, described tests with a large variety of chemicals which have been tried as molluscicides. Of some 135 tested only three, namely, dinitro-o-cyclohexylphenol, 2-4-dinitro-6-phenylphenol, and pentachlorophenol, showed promise of being effective. Nothing substantially more efficient than copper sulphate has emerged. Some interesting ecological work indicated that snails prefer areas high in calcium and phosphorus with a *p*H of 7·5 to 8·5 and supplied with water of subterrestrial origin or which has been flowing over heavily limed soils.

The papers in the section on small animals comprised the following:—

A symposium on fractures with demonstrations of the Thomas splint, the Schaffer sleeve, and internal fixation appliances; a discussion on pre-anaesthetics, curare, analeptics, etc., illustrated by a film; the diagnosis, incidence, etc., of histoplasmosis; the diagnosis and prognosis of foreleg paralysis in the dog; the use of a plasma extender in animals; and the combining of a large- and small-animal practice in the same premises.

In discussing pre-anaesthetics, Dr. Kingma, of Columbus, Ohio, stated that many U.S. veterinarians tend to dispense with these on the plea of insufficient time. In his opinion this is unwise in

nervous, worried or excited animals which fight restraint and the induction of anaesthesia and which, because of this, often are poor operation risks. For dogs, morphine and atropine given about three-quarters of an hour before operation are used. The depression of the respiratory centre caused by morphine passes off within half an hour and the action of atropine on the vagus nerve gives a high degree of safety. For induction of anaesthesia Dr. Kingma employs pentothal sodium which he follows with chloroform and curare for maintenance. He regards curare and mephenesin, another curare-like compound, as very useful adjuncts in veterinary anaesthesia.

Dr. Benjamin, a member of the medical profession, of Milwaukee, Wisconsin, described excellent results which he had obtained in dogs with a plasma substitute known as okra plasma. Transfusions after experimental haemorrhage into a state of shock with blood pressures down to as low as 0 mm. of mercury and cessation of breathing for as long as three minutes were uniformly successful. In various disease conditions the plasma substitute gave good results.

As might be expected in view of the huge poultry industry in the United States, the poultry section was very active. In talking on "Disease and the Poultry Industry," Dr. Carpenter, of Chicago, Illinois, made the point that each U.S. veterinarian was now responsible for the health of no less than £150,000 of poultry. The industry, which is now the largest in the world (Americans eat on the average 406 eggs per head each year), continues to expand. The population of 480 million laying hens was at one time larger than that of the table birds or broilers but these have now far outstripped it. Some idea of the expansion which has taken place and its rapidity during the last few years is given by the fact that the broiler population which was 100 million in 1939 had multiplied to 450 million by 1949, increased to 616 million in 1950 and during the present year is expected to exceed 725 million head. At an average of 3 lb. of poultry meat per bird and with very high cattle prices, poultry meat is now becoming the equal of beef in the national diet.

In addition to Dr. Carpenter's paper, contributions covered the following subjects: Diagnosis and control of respiratory diseases; prevention and control of bacterial diseases; biological products in poultry diseases; and the laboratory as an aid to the poultry practitioner. In addition, one discussion group dealt with bacterial diseases, and another with respiratory disorders.

The section on surgery and obstetrics covered a wide field with most emphasis on gynaecological subjects. A film was shown dealing with the functional anatomy of the reproductive tract of the bovine and there were panels on reduced fertility in cattle, maintaining fertility in the bull, and aids in dystokia. There were papers, also, on caesarean section in the cow, perineorrhaphy for correction of relaxed vagina, the level of progesterone necessary to maintain pregnancy in dairy cattle, and the indications for caesarean section in the bitch. The session also included a very interesting paper by Dr. Nichols, of Wisconsin University, Madison, on the physiology of the rumen in bloat.

In the panel on reduced fertility in cows, papers were presented on bovine endometrium infections, fertilisation and embryonic death in dairy cattle, and hormone therapy. An interesting point which emerged is that general field experience in the United States is that in anoestrus about 90 per cent. of ovaries examined prove to be normal and in these cases hormone treatment does more harm than good. Uterine and cervical infections are held to be the main cause of infertility.

In dealing with bloat, Dr. Nichols made a plea for reconsideration of the mechanical factors of function of the rumen in this connection. The degree of fill may in his estimation be of prime importance. Dr. Nichols has been able to show experimentally that an artificially produced gas pressure following overloading of the rumen with water will rapidly produce death. Dr. Pounden, of Beltsville, Maryland, in discussing Dr. Nichols's paper, brought out the point that all the literature on bloat stresses the presence of water or herbage with high moisture content. He considers that certain herbage may have an osmotic pressure which draws fluid into the rumen and renders the animal unable to deal with gas.

In the section on public health the papers dealt mainly with the objectives of public health and animal disease control, present and future animal-human health problems, the co-ordination of veterinary public health and animal disease control problems, veterinary public health in a city health department, the functional rôles of the veterinarian and the sanitary engineer in public health, the future outlook towards special weapons of defence, and disease control with special reference to brucellosis. In addition, there were panels on veterinary public health at work and on the importance of veterinary public health in civil defence.

Closed Circuit Television

Undoubtedly the greatest interest at the Convention was shown in the closed circuit television. This was the first occasion on which this means of demonstration had been used before a veterinary audience and it was clearly a great success. The rooms below the main auditorium had been converted into studios and operating theatres and the broadcasts (or telecasts as they were called) were made on two large screens about the size of those employed in small cinemas. These had been set up at one end of the hall. At each session a selected commentator described the operations step by step while full details were given through close-up and long-distance camera shots. The arrangements for the programme were made jointly by the Radio Corporation of America and Allied Laboratories Inc. and were very well carried out. There were five sessions in all, dealing respectively with anaesthesia (one hour), conducting a poultry practice (30 minutes), laboratory procedures in disease diagnosis (30 minutes), canine surgical demonstrations (one hour), and large-animal surgical demonstrations (one hour). It was an impressive experience to sit with some 3,000 other people and in considerable comfort, to watch demonstrations taking place, every detail of which easily could be seen.

The narrator in the first session on anaesthesia was Dr. Farquharson, Professor of Surgery at the Colorado Veterinary School, who, many will recall, provided some of the highlights of the 14th International Veterinary Congress in London with his films and commentaries on surgical subjects.

In the horse, epidural anaesthesia with procaine was demonstrated, followed by various nerve blocks employing procaine with hyaluronidase, the so-called spreading factor. General anaesthesia of a colt with "Nembutal" was attempted but was not very successful.

Paravertebral anaesthesia in the bovine was then demonstrated using "Pontocaine" with adrenaline. Nerve block of the eye and associated structures was also shown.

In the pig, Dr. Hardenbrook, of Urbana, Illinois, demonstrated the technique of epidural anaesthesia using the lumbo-sacral space and injecting 1 c.c. of procaine, 2 per cent. solution per 10 lb. bodyweight. General anaesthesia with "Pentothal" was also shown.

In the small-animal part of the session for which Dr. Knowles, of Miami, Florida, was narrator, anaesthesia in the dog was induced by endotracheal tube, and rebreathing bag apparatus. This type of anaesthesia is employed for canine thoracic surgery. The demonstration also featured the post-operative use of an oxygen tent. In the cat a remarkable demonstration of handling and restraint was given by Dr. Moe, of Hammond, Indiana.

The second and third television sessions dealt with the poultry field and with public health. Dr. Nelson, of Jewell, Iowa, and Dr. Lee, of Ames, Iowa, in a studio fitted up to resemble a veterinarian's office with a *post-mortem* area on one side, demonstrated how to diagnose and advise on the various poultry diseases which the average practitioner may be expected to encounter. Following this a comprehensive demonstration was given of laboratory procedures in general disease diagnosis. This included the ring test for brucella agglutinins in milk, procedures for mastitis diagnosis, the haemagglutination and haemagglutination-inhibition tests for Newcastle disease, the procedure for rabies examination, and a technique for staining bull semen.

Dr. Schlotthauer, of the Mayo Foundation, acted as narrator for the canine surgical demonstrations. These comprised open reduction and intramedullary pinning of a femoral fracture in a cat, excision of the patella in a dog, and the removal of a foreign body from the stomach of a dog. The intramedullary pinning

was accomplished very rapidly and precisely, the whole operation from start to finish taking just 13 minutes. Patellectomy was stated to be performed quite frequently and with success in the dog when lameness is due to luxation of the patella, or in fractures, etc. The ends of the severed ligaments are sutured and the leg immobilised in splints for two to three weeks.

Two methods were attempted for removal of a foreign body (a beer-bottle cap) from the stomach of a dog. The first was removal by a gastroscope following laparotomy. This was unsuccessful chiefly owing to the stomach being full of food. Gastrotomy had, therefore, to be employed.

In the final television session two very interesting large-animal surgical demonstrations were given. These were commented upon by Dr. Frick, Professor of Surgery, Kansas Veterinary School. They comprised caesarean section in the pig and rumenotomy in a bovine for traumatic gastritis. The latter operation was undertaken under paravertebral anaesthesia and was conducted at high speed. The muscle layers were cut through by a single stroke and following the removal of the foreign body (a bottle) the wound was closed with single figure-8 sutures of umbilical tape which enclosed peritoneum, muscle and skin in one movement. It was stressed that cleanliness and good technique were essential for good results, but that these need not slow down the speed of the operation unduly.

The Convention closed at lunch-time on Thursday after a simple ceremony during which the new President was inducted. With few exceptions contributions were very well presented with ample illustrative material and adequate detail, and an excellent balance was maintained throughout between visual matter and the spoken word.

One could not help being impressed with the energy and enthusiasm which had been shown throughout by speakers and audience alike, despite room temperatures of over 90° F. and high humidity. Technically, American veterinary science, as judged by most of the contributions, is at a high level.

On a personal basis it is a pleasure to record the friendliness and co-operative spirit which everywhere prevailed. It would surely be difficult to find anywhere where one could make so many friends in so short a time. As travel becomes easier and distances shorter it can only be hoped that there will be a much greater exchange of veterinarians between the two countries than there is at present. One would like to feel that everything possible is being done to this end. We in Britain now have much to learn from America, which has the most powerful and, possibly, the most progressive veterinary profession in the world. The Americans, too, can, I think, still learn something from us and are very willing to do so. I am sure that it would be to the lasting benefit of both countries and both professions if the N.V.M.A. and the A.V.M.A. together could work out some scheme whereby an exchange of speaker-members was arranged each year from both sides. Such a scheme would certainly have the support of our American colleagues.

D. D. Ogilvie.

LIMITING LEAD IN FOODS: STANDARDS PROPOSED

The introduction of limits for lead in food and drink are recommended in a report presented to the Food Standards Committee by its metallic contamination sub-committee.

In the report—approved for publication by the Minister of Food—the sub-committee points out that lead is a cumulative poison so that repeated small doses, each of which is too small to produce any demonstrable effect by itself, may in course of time prove toxic. Complete elimination of lead from food and drink is not immediately possible.

The analytical data collected by industries is incomplete and the method of lead determination is complicated, the report states. Time is needed for the elimination of lead pipes and containers, the search for lead substitutes, and the revision of certain factory processes, as well as for the development of simpler analytical methods of lead determination, and for analytical surveys. In the meantime it is proposed that an industrial committee should be set up to advise on the technical problems involved, to ascertain whether and to what extent the limits recommended in the report are being exceeded and to review progress from time to time.

University of Edinburgh Royal (Dick) School of Veterinary Studies

WAR MEMORIAL UNVEILING CEREMONY

On Tuesday, November 27th, 1951, in the War Memorial Library of the Royal (Dick) School of Veterinary Studies, the unveiling ceremony of the War Memorial to the former students of the Royal (Dick) Veterinary College who gave their lives in the 1939-45 war took place.

Sir Edward V. Appleton, G.B.E., K.C.B., M.A., D.Sc., Sc.D., LL.D., F.R.S., Principal and Vice-Chancellor of the University of Edinburgh, presided over a representative gathering, including the staff and students of the School and relatives and friends of the fallen, in the hall, where, after a prayer of intercession by the Rev. Robert Taylor, M.A., Mr. W. M. Mitchell, M.C., T.D., M.B., Ch.B., B.Sc., M.R.C.V.S., F.R.S.E., Director of Veterinary Education, invited the Principal to accept custody of the Memorial on behalf of the University. This the Principal did, in the following address :—

> "It is with mixed feelings—and yet feelings in which pride is uppermost—that I accept from the Director of Veterinary Education, on behalf of the University, the Memorial to members of the Royal (Dick) Veterinary College who died for their country in the last world conflict. These members of the College were men who were first drawn, by a sense of vocation, to devote their lives to veterinary work—to the art and science of healing—and this alone would commend them to us. But more than that, when danger menaced our islands, they saw the path of duty clear and unmistakable before them, and pursued it.
>
> "Your sense of loss, when they failed to return, is, we know, deep and permanent. The University, in which is now incorporated the College they knew and loved so well, is also proud to honour their memory and their sacrifice. Among their friends and fellows are many whose names are inscribed on the Memorial in the Old College; and, on behalf of the University, I can pay no more fitting tribute to *your* sons than to remember them, proudly and gratefully, among those sons of *ours* who 'turned without fear or question from these Gates of Learning to those of the Grave, in order that free men should still continue to learn freedom.'
>
> "Director, in accepting this Memorial, I give you the simple assurance that we shall cherish it and treasure it for all time."

Mr. Mitchell then expressed his regret that the accommodation in the War Memorial Library was not sufficient to allow of everyone being present at the unveiling ceremony, but he invited the relatives and friends, and representatives of the staff and students, to join him there, and explained that the ceremony would be relayed to the hall, so that those who could not see would hear, and be with them in spirit.

In the Library, the ceremony was opened by the Rev. David H. C. Read, M.A., B.D., University Chaplain, in a prayer of thanksgiving. The Principal then invited Mrs. Helen Steele, the widow of Major A. P. Steele, I.C. (R.A.), B.Sc., M.R.C.V.S., D.V.S.M., one of the former students commemorated on the Memorial, to perform the unveiling ceremony.

This was followed by a prayer of dedication by Mr. Read, and after the Benediction wreaths were laid on behalf of the following :—

The Royal (Dick) Veterinary College Alumnus Association, by the President, Mr. J. N. Ritchie, B.Sc., M.R.C.V.S., D.V.S.M. ;

The Director and Officers, Army Veterinary and Remount Services, by Major G. D. Young, M.B.E., M.R.C.V.S., R.A.V.C. ;

The Staff and Students of the Royal (Dick) School of Veterinary Studies, by Mr. W. M. Mitchell.

The ceremony closed with the sounding of "The Last Post" and "Reveille."

The Memorial takes the form of a list of 23 names added to the 1914-18 War Memorial Tablet in the War Memorial Library of the School, and is inscribed as follows :—

PRO PATRIA

1914-1918

Jas. S. H. Bennett	Thomas Brown
J. W. Brownless	W. Tully-Christie
David Hannay	George Hislop
Henry C. Lowry	Donald McCallum
Max Mendelsohn	Ian Ness
D. S. Phorson	Jacob H. Primmer
John Storie	E. L. Tottenham

1939-1945

D. D. P. Bowie	I. M. Brown
R. D. Brown	K. G. Comrie
A. M. Craigen	C. Don
R. Dunwoody	J. Forsyth
S. J. Gillmor	G. D. Graham
J. A. Hawkins	J. Lang
A. G. McCrea	K. D. McDougall
L. T. Morris	H. J. Parker
C. B. Pearson	G. Rutherford
A. P. Steele	W. E. Stenhouse
J. W. Urquhart	L. Warren-Smith

G. Wharton

ROYAL COLLEGE OF VETERINARY SURGEONS

Membership Examination, December, 1951

List of Successful Candidates

London:

First Examination

Both subjects:

Blaxland, D. R.

One subject:

Carter, P. A.	Hudson, M. R.	Underdown, J. W.
Graham, J. M. H.	Rutherford, L. H. (C)	Wilkinson, John

(C) denotes credit in Chemistry and Physics.

Second Examination

Anatomy only

Beaumont, S. H.	Dubery, B. R. E.	Keeling, M. D.
Brazier, J. F. H.	Fenton, B. K.	Knight, S. A.
Cullen, C. S.	Gee, R. E.	Norman, D. A.
Davidson, H. M.	Hardwick, A. G.	Pigott, R. C. C.
Davies, M. G.	Jones, R. H.	Vickers, G. E.

(To be continued)

QUESTIONS IN PARLIAMENT

Veterinary Surgeons, Wales

Mr. Watkins (December 4th) asked the Minister of Agriculture how many veterinary surgeons are located in each of the counties of Wales; and whether he is satisfied that there are sufficient available for the counties of Brecon and Radnor.

Sir T. Dugdale: The number of veterinary surgeons in general practice resident in each county in Wales is as follows:—

Anglesey	6
Brecknockshire	6
Cardiganshire	17
Carmarthenshire	31
Caernarvonshire	12
Denbighshire	19
Flintshire	9
Glamorgan	33
Merionethshire	5
Monmouthshire	16
Montgomeryshire	11
Pembrokeshire	16
Radnorshire	1
	182

Besides those in private practice there are 50 veterinary surgeons in Wales including 45 in the Ministry's services, holding full-time veterinary appointments.

There is a general shortage of veterinary surgeons and I am not aware that there is more difficulty in obtaining their services in Brecon and Radnor than elsewhere.

Monkeys (Importation)

Lieut.-Colonel Lockwood asked the Minister of Agriculture whether, in view of the large numbers of monkeys now being imported by air, he will make the necessary Regulations to prevent suffering through overcrowding.

Sir T. Dugdale: The Protection of Animals Act, 1911, makes it an offence to convey or carry any animal in such manner as to cause that animal any unnecessary suffering. There would appear to be no need to make a Regulation to cover the particular case where suffering is due to overcrowding.

Damage by Animals (Legal Liability, Report)

Mr. Janner (December 3rd) asked the Attorney-General when the committee appointed to consider the law of civil liability for damage done by animals is expected to issue its report.

The Attorney-General: The Committee only commenced its examination of this very complex subject in October and it is too early to forecast the date of its report.

Duck Eggs (Infection)

Mr. Janner (November 26th) asked the Minister of Food whether he is aware that duck eggs may be poisonous unless boiled at least 10 minutes and should be eaten within three or four days unless kept in refrigerators; and whether he will arrange that printed cards containing these instructions be placed prominently on the counters of all shops where duck eggs are sold.

Dr. Hill: There is some slight risk of infection which makes it desirable to cook duck eggs thoroughly. This point receives wide publicity and I do not think we need special placards on the subject.

New Cars (Waiting List)

Miss Elaine Burton (November 26th) asked the Minister of Supply if he will take powers to ensure that people whose names have been on a waiting list many years have priority in the delivery of new cars.

Mr. Sandys: No, Madam.

Miss Burton: In view of the widespread dissatisfaction throughout the country on this matter, may I ask if the right hon. Gentleman is aware of the racket, which is perfectly legal, by which car dealers take cars from customers at around two years old, and therefore outside the covenant, and sell them at twice the listed price, and so are able to make that profit plus the profit of a new car, and that that means that people on the normal waiting list have just no chance at all of their priority?

Mr. Sandys: It is a very difficult problem, but I do not think it is one which the Government can tackle. During the war, when there were comparatively few private cars being produced, it was possible to control the priorities for those cars. We now have to deal with about 80,000 cars a year. It is quite beyond the organisation that we could manage to set up—we should not be justified in setting up a larger one—to try to decide who should and who should not buy these cars.

Mr. G. R. Howard: Cannot my right hon. Friend consider the policy of releasing in the next few months a larger number of cars to the home market so that dealers can see what is the real position of the waiting list and remove many who have their names down for two or three different cars?

Mr. Sandys: We should very much like to increase the number of cars available to the home market, but we have to bear in mind the importance of the export trade at this moment.

Miss Burton: I appreciate that it is beyond the powers of the present Government to deal with this, but will the right hon. Gentleman do something to ensure that ordinary people do get the priorities to which they are entitled?

Mr. Sandys: As I explained to the hon. Lady last week, the industry are doing their best to ensure that specially urgent categories of people, such as doctors, district nurses, and midwives, do receive priority. If there should be other categories of people for whom it is urgent and important to have new cars, I am quite prepared to make representations to the industry to see that they get special attention.

Gambia Poultry Scheme (Report)

Mr. J. Johnson (December 6th) asked the Secretary of State for the Colonies when he hopes to publish the report of the Commission upon the Gambia Poultry Scheme.

Mr. Lennox-Boyd: I cannot yet say. The enquiry is still in progress and in accordance with the Commission's terms of reference, the question of publication will be determined only after the report has been received and considered by His Majesty's Government.

Livestock (Spray Treatment, Trials)

Mr. Hurd (December 6th) asked the Minister of Agriculture what progress has been made with trials to test the efficacy in this country of the spray treatment of sheep rather than dipping as a method of destroying animal parasites.

Sir T. Dugdale: Trials have been made this year by a commercial firm with the "deluge" type of sprayer, under field conditions, and some success in fly control has been achieved. We are anxious to do some work on the use of sprays for external parasites of livestock but the resources of my Department's veterinary laboratory at Weybridge are limited, and it has not yet been possible to find time for this work.

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Sheep Worrying

Mr. G. Williams (December 6th) asked the Minister of Agriculture what discussions he has held with the National Farmers' Union on the protection of sheep from killer dogs; and what conclusions have been reached.

Sir T. Dugdale: A preliminary discussion between officers of my Department and the National Farmers' Union took place a short time ago. I am still considering the proposals put forward by the Union.

NOTES AND NEWS

Diary of Events

Dec. 19th.—Meeting of the Lancashire Division, N.V.M.A., at Liverpool (School of Tropical Medicine, Pembroke Place), 2.30 p.m.

Dec. 21st.—Council Meeting of the Society of Practising Veterinary Surgeons Division, N.V.M.A., at 36, Gordon Square, London, W.C.1, 2.30 p.m.

Jan. 4th.—Meeting of the Association of Veterinary Teachers and Research Workers (Scottish Branch), in the University of Glasgow Veterinary School (83, Buccleuch Street), 9.30 a.m.

Jan. 9th.—Meeting of the Association of Veterinary Teachers and Research Workers (Southern Region), at the Royal Veterinary College, Camden Town, N.W.1.

Jan. 9th, 10th and 11th.—Meeting of Committees and Council, R.C.V.S., at 10, Red Lion Square, London, W.C.1.

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PROFESSIONAL INCOMES

An organisation which needs figures showing the increases which have taken place in incomes in other professions since 1939 has sought the help of the N.V.M.A.

It requires, in particular, information regarding incomes from general practice and preferably covering overall earnings.

Being desirous of giving the organisation in question every assistance, the N.V.M.A. invites the following particulars from those members of the Association who have been in general practice as principals during these several years:—

(i) Figures showing the percentage of net "profit" in relation to gross turnover for 1939.

(ii) Figures showing the percentage increase in the gross turnover for each full year since the war (*i.e.*, 1946-50), showing 1939 as 100 per cent.

(iii) Figures showing percentage of net "profit" for each full year since the war in relation to gross turnover.

As an example of the kind of information required the following case (fictitious) is given:—

1939.—Gross turnover (£1,000) (not to be disclosed).

Net profit (£100) „ „ „

Therefore proportion of profit to turnover is 10 per cent.

1946.—Gross turnover (£2,000) (not to be disclosed).

Therefore gross turnover in relation to 1939 is 200 per cent.

Net profit (£200) (not to be disclosed).

Therefore proportion of profit to turnover is 10 per cent.

It is requested that replies should be sent to the General Secretary, N.V.M.A., 36, Gordon Square, London, W.C.1, by *December 14th.*

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PERSONAL

Forthcoming Marriage.—Pfob—Robertson.—The engagement is announced between Major Lewis S. Pfob, M.R.C.V.S., Uganda Veterinary Service, only son of Mr. and Mrs. S. Pfob, Coral Mount, Kirkinti loch, and Moira, younger daughter of Mr. and Mrs. Wm. Robertson, 36, Ripon Drive, Glasgow.

Marriage.—Pink—Ayhan.—On November 21st, 1951, at Tel Mond, Israel, Cyril E. Pink (Varod), B.V.Sc., M.R.C.V.S., youngest son of Mr. and Mrs. Charles Pink, Liverpool, to Edith Ayhan, daughter of Mrs. S. Ayhan, Istanbul.

Rayne—Coate.—On November 24th, 1951, at All Saints' Church, Langport, Somerset, Philip Rayne, only son of Mr. and Mrs. J. E. Rayne, of Plymstock, South Devon, to Pamela Mary Coate, only daughter of Mr. and Mrs. C. H. Coate, of Langport, Somerset. Mr. P. N. Brotherton, M.R.C.V.S., was best man.

Richardson—Franks.—On Tuesday, December 4th, 1951, at St. Mary's, Merton, John Richardson, M.R.C.V.S., only son of Mr. and Mrs. R. G. Richardson, of New Malden, Surrey, to Gweneth Mary, only daughter of Mr. H. C. Franks and the late Mrs. H. C. Franks, of Merton, Surrey.

Mr. W. G. Beaton's Appointment as Director of Inter-African Bureau of Epizootic Diseases

Mr. W. G. Beaton, M.R.C.V.S., D.T.V.M., retiring Director of Veterinary Services, Nigeria, has been appointed to be Director of the Inter-African Bureau of Epizootic Diseases to be set up in Kenya at the site of the East African Veterinary Research Organisation at Mugaga, near Nairobi. The bureau has been established under the auspices of the Commission for Technical Co-operation in Africa south of the Sahara (C.C.T.A.).

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FOOT-AND-MOUTH DISEASE CONTROL

N.F.U. Statement in Support of Slaughter Policy

On Saturday last the National Farmers' Union issued a statement in which the opinion was expressed that slaughter was the only reliable method of controlling foot-and-mouth disease. Some advance had been made in the preparation and use of vaccine, but that method was not foolproof and was subject to severe limitations.

No effective alternative to slaughter had yet been found which would give immunity to our stock and maintain the confidence of the world's buyers in the reputation of British livestock. For that reason most farmers represented by the National Farmers' Union had consistently supported the Ministry of Agriculture in maintaining the present policy of slaughter to save the livestock industry from ruin.

The use of vaccine in some Continental countries, where the disease was rampant, was not a valid argument for its adoption in this country which, as an island, generally enjoyed much more freedom from land-borne infection than the Continent.

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FOWL PEST: CONTINUANCE OF POULTRY IMPORT BAN

The previous Government decided earlier in the year that imports of poultry from countries where fowl pest is endemic should be prohibited in view of the serious risks involved for our own poultry industry. It was also decided that the position should be reviewed later in the year.

This review has now taken place and the Government has decided that the general prohibition must be continued. It has been agreed, however, that imports will be admitted from a specified area in France subject to certain conditions designed to ensure that there is no risk of bringing infection into this country.

Outbreaks of fowl pest in recent weeks have been practically confined to an area in Lancashire. The Ministry of Agriculture considers that there is a good chance that it will be possible to eradicate the disease provided that the import prohibition is continued and that poultry farmers continue to co-operate by reporting any symptoms of disease immediately they are seen.

When the ban on imports was announced last spring, the Minister of Agriculture appealed to the home poultry industry to rear more birds for the table so as to fill the gap left by the prohibited imports. The industry has responded well and it is expected that total supplies of turkeys and chickens will be only slightly below what they were last Christmas.

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THE ANIMAL HEALTH TRUST

The Duke of Norfolk, President of the Animal Health Trust, has announced that in order to consolidate and extend its programme of research into the diseases of livestock, the Executive Committee, under the Chairmanship of Mr. J. Arthur Rank, has been confirmed in office by the Trustees and Council and becomes the Trust's permanent Committee of Management. The Committee, in addition to its Chairman, consists of:—

Sir Richard Haddon; Mr. Cole Tinsley, Vice-Chairman of the National Farmers' Union; Mr. J. K. Knowles, General Secretary of the National Farmers' Union; Lord Stamp; Mr. R. Bolton, Past-Chairman of the Farmers' Club; Dr. W. R. Wooldridge, Scientific Director and Chairman of Council, Animal Health Trust.

This announcement was made following the meeting of the Trustees and Council held on November 29th, when in thanking them for their invaluable work, the Duke of Norfolk added that as a result of helpful and constructive discussion with the Ministry of Agriculture and Fisheries, and The Agricultural Research Council, the Committee was now at work consolidating the Trust's management and finances on a permanent basis.

At the meeting it was announced also that, as a gesture, the Trust had invited an independent firm of Chartered Accountants to report upon its general management and administration. The report had been wholly favourable

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ADDRESSES OF DISEASE-INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

ANTHRAX:

Cheshire.—Moor Farm, Shocklach, Malpas (Dec. 1st); Heathfield Farm, Dean Row Road, Wilmslow, Manchester (Dec. 3rd).

Denbighshire.—Sun Inn, Sun Bank, Llangollen (Dec. 7th).

Kirkcudbrightshire.—Low Arkland, Bridge of Die, Castle Douglas (Dec. 4th).

Lancs.—Town Head Farm, Grindleton, Clitheroe (Dec. 5th); Catshaw Hall Farm, Over-Wyresdale, Bay Horse, and Lower House, Wray, Lancaster (Dec. 7th).

Monmouthshire.—Prescold Camp, Coed-y-Paen, Nr. Pontypool (Dec. 7th).

Northants.—Benbow Field, Newton Bromswold, Northants (Dec. 1st).

Wilts.—Whyr Farm, Potterne, Devizes; Eyres Farm, Heddington, Calne (Dec. 3rd).

Worcs.—Old Castle Farm, Colwall, Malvern (Dec. 5th).

FOOT-AND-MOUTH DISEASE:

Essex.—Park Farm, St. Osyth, Clacton-on-Sea (Nov. 30th).

Berks.—Foliejon Park Farm, Winkfield, Windsor (Dec. 2nd).

Lincs.—Agarth Farm, Legbourne Road, Louth (Dec. 4th).

Norfolk.—Half Piece Field, Middle Farm, Colney, Norwich (Dec. 1st); Thorpe Hall, Thorpe, Haddiscoe, Norwich; Old Oak Farm, Hopton, Gt. Yarmouth; Morley Manor, Wymondham (Dec. 3rd); Hall Farm, Thrigby, Gt. Yarmouth (Dec. 4th); Grove Farm, Rollesby, Gt. Yarmouth (Dec. 6th); Mill Farm, Tharston, Long Stratton, Norwich; Heath Farm, Caister, Gt. Yarmouth (Dec. 7th); Hall Farm, Scrathy, Gt. Yarmouth (Dec. 8th).

Suffolk (E.).—Wood Barn Farm, Mutford, Beccles; Valley Farm, London Road, Beccles (Dec. 5th); Lodge Farm, Shadingfield, Beccles (Dec. 6th); Ashby Hall Farm, Ashby, Lowestoft (Dec. 9th).

Yorks (E.R.).—New Farm, Plaxton Bridge, Thearne, Beverley (Dec. 1st); Manor Farm, Grindale, Bridlington (Dec. 3rd); West Birmington Farm, Staxton Scarborough (Dec. 7th); Willerby Grange Wold Farm, Willerby, Scarborough (Dec. 9th).

FOWL PEST:

Essex.—Betts Farm, Saling, Braintree (Dec. 6th).

Lancs.—All at Preston: Primrose Farm, Hoole; Lyndene, Liverpool Old Road, Much Hoole; Peel View Poultry Farm, Drumacre Lane, Longton; Brook Farm and Southlands, Lytham Road, Warton (Nov. 30th); Myrtle House, Lytham Road, Freckleton (Dec. 1st); The Dolphin Inn, Marsh Lane, Longton (Dec. 1st); Longcroft and 20, Gill Lane, Longton; Moss House Farm, Moss Lane, Little Hoole; Briar Field and The Lodge, Liverpool Old Road, Hoole; Briarfield, Liverpool Road, Hutton (Dec. 4th); Field opposite Primrose Cottage, also Hazel Dean and Stanley Dene, Station Road, Hoole; Lorwain, Station Road, Little Hoole; Back Lane, Longton (Dec. 5th); Bank Farm and Whinfield, Station Road, Little Hoole; Weaver's Farm, Grange Lane, Hutton; Stanley Farm, Rose Acre, Kirkham (Dec. 6th); Weaver's Fold, Bretherton; Laurel Bank, Hallcar Lane (Dec. 7th); Rushmoor, Shirley Lane, Longton (Dec. 8th); Marsh House, Station Road, Little Hoole (Dec. 7th).

Suffolk (W.).—Genfou, Colchester Green, Cockfield, Bury St. Edmunds (Nov. 30th).

SWINE FEVER:

Angus.—Mains of Fintry Farm, Dundee (Dec. 3rd).

Denbigh.—1, High Street, Giversyllt, Wrexham (Dec. 5th).

Essex.—Berechurch Poultry Farm, Kingsford, Colchester (Dec. 3rd); Waylett's Café, Arterial Road, Dunston (Dec. 6th).

Herts.—Walnut Tree Farm, Piton, Hitchin (Dec. 3rd).

Hunts.—Dundee House, Broadway, Yaxley (Dec. 4th).

I. of Ely.—Stitches Farm, Wimblington (Dec. 4th); Downham Road, Ely, and Alderforth House, Main Street, Witchford, Ely (Dec. 6th).

Kent.—White Post, Preston, Canterbury (Dec. 7th).

Leics.—Ingle Pingle, Forest Road, Loughborough (Dec. 3rd).

Lincs (Kesteven).—White House Farm, Timberland (Dec. 6th); Metheringham Field Farm, Martin Moor, Martin (Dec. 8th).

Lincs (Lindsey).—The Orchard, Little Lane, South (Dec. 3rd); Poplar Farm, Grainthorpe (Dec. 5th).

Norfolk.—Weybourne Road Allotments, Sheringham (Dec. 7th).

Northants.—2, Woodcock Street, Burton, Latimer (Dec. 7th); Glebe Farm, Cranford St. John, Kettering (Dec. 7th).

Oxon.—Moor End Lane Farm, Thame (Dec. 4th).

Salop.—Platt Farm, Prees (Dec. 8th).

Staffs.—Highgreaves Lodge, Whitmore Road, Clayton; Burgess Field, Albemarle Road, Newcastle (Dec. 3rd).

Wilts.—Fair View, Hawk Street, Bromham, Chippenham (Dec. 3rd).

Worcs.—Dripping Well Farm, Wribbenhall (Dec. 4th); The Hill, Upper Moore, Pershore (Dec. 5th).

Yorks (W.R.).—Height Farm, Delph Oldham (Dec. 5th).

ALLEGED DOPING OF RACEHORSES

As our readers will be aware, investigations are being made into the alleged drugging of racehorses, and much public interest has been aroused by Lord Rosebery's assertion that one of his animals has been doped and his offer of a reward of £1,000 for information which might establish the identity of those responsible. The enquiries which Scotland Yard have been conducting for several months have culminated in a conference of officers concerned with racing irregularities and it is understood that the Jockey Club is to hold an emergency meeting.

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ERRATA

Mr. J. F. MICHEL, of the Veterinary Laboratory of the Ministry of Agriculture and Fisheries, sends the following correction of the report of his contribution to the discussion following on "Notes on Husk" in our issue of November 10th (p. 706, col. 1, para. ii):—"What I did in fact say was that resistance to husk may be regarded as taking the form of two separate local mechanisms in the mesenteric lymph glands and in the lungs. In the resistant bovine few worms appear to get past the lymph glands and reach the lungs, and those that do develop only very slowly, their rate of growth bearing an inverse relation to the level of complement fixing antibody."

And Mr W. ALLCROFT, Provincial Poultry Advisory Officer of the Ministry, writes:—"In the interests of accuracy, may I point out that the article by W. O. Brown, of which you published an abstract on page 724 of your issue of November 17th, appeared in the Journal of the *Science* of Food and Agriculture, and not as at the bottom of the abstract." (*J. Sci. Food and Agric.*).

Crookes Halibut Oil (Veterinary).—The Crookes Laboratories Limited write to point out that at the time the information was given us, upon which was based our reference to the vitamin values of their halibut oil product, appearing in the description of their exhibit in our Congress Number, the firm did not, in fact, issue a special Veterinary brand, and the figures quoted were for the standard oil for human consumption. However, Crookes do now manufacture a special brand of Crookes Halibut Oil (Veterinary) which provides the primary requirements for veterinary work so far as the two most important oil soluble vitamins are concerned, specified as 10,000 International Units of A and 2,000 of D per gramme, at a low price. The product is available in ¼ gallon and 1 gallon tins.

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the N.V.M.A.

HARD PAD DISEASE

Sir,—Ever since hard pad was first recognised there have been several communications to *The Veterinary Record* on the subject of "cures" and treatment. Whilst all practitioners welcome suggestions for treating this disease, I am far from satisfied that the "cures," "recoveries" and "absence of nervous sequelae" claimed can be attributed to the drug or drugs used.

As with most virus diseases, considerable variations occur in mortality and symptomatology in different outbreaks. There seems to be no doubt that the natural recovery rate in many outbreaks of hard pad is quite high, and one is often surprised that dogs that have been ill for several weeks recover without any proper treatment. By virtue of the close relationship between the viruses of distemper and hard pad, dogs that have been actively immunised or that have acquired a natural immunity to distemper have also a partial or complete immunity to hard pad. It is therefore not uncommon to get a high recovery rate in many outbreaks or an absence of nervous symptoms. Bearing in mind this natural recovery rate, I fail to see how either Mr. Toosey or Miss Milnes can

claim that the drugs they have used give "good results" and a "recovery rate of 73 per cent." respectively.

As far as is known, chloromycetin has no anti-viral action, but is active against Gram-negative organisms present as secondary invaders. At the moment the only specific anti-viral agent known is hard pad serum and the value of this serum as a prophylactic and curative has been discussed elsewhere. It is also noted that in these articles on the treatment of hard pad there is a tendency to use several drugs at the same time in an effort to allay the symptoms. Very often these drugs are contra-indicated as the dog is a ready toxic, due to physiological disfunction, *e.g.*, myocarditis, enteritis, pulmonary oedema, etc., and their use may hasten the death of the dog.

Until it is proved that a drug is active against the virus of hard pad and can cut short the duration or course of the disease we should not use the terms "cures" and "recovery rates" but rather say "drug X has been used with apparently beneficial results."—Yours faithfully, G. E. Joss, 6, Earlswood Road, Redhill, Surrey. *December 1st*, 1951.

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CURARIZING DRUGS

Sir,—As the individual solely responsible for the original research on Myanesin; may I crave a few lines of your space in which to try to correct certain misconceptions put forward by some of my esteemed veterinary colleagues?

Miss Phillips asks about dosage and toxicity. The effective dosage in man is most scientifically expressed as 10 to 15 mg. per kg., but 25 to 30 mg. per kg. is well within safe limits in cats (Berger & Bradley, 1946). In human subjects the recovery time from anaesthesia is not affected by Myanesin except in as much as the reduced dose of pentothal required results in a quicker regaining of consciousness.

I really must gently chide my friend Mrs. Sheppard (*Vet. Rec.*, 1951) for the statement that Myanesin acts on the myoneural junction. I have been unable to find any published paper suggesting this site of action and I have certainly never suggested it to her myself. The comprehensive account of the pharmacology of Myanesin by Berger and Bradley (*op cit.*) definitely points to the spinal cord as the principal site of action of the drug.

Mr. Longley and Mr. Cowie (*Vet. Rec.* **63**, 745 & 716) I fear did not read my paper of 1947 (Mallinson, 1947) very closely; otherwise they would have noted that I made it clear that narcosis in animals is produced only by enormous doses of Myanesin, doses in the region of 200 to 300 mg. per kg. and doses which are vastly in excess of those ever used clinically. Mr. Longley will find if he cares to experiment (as did Berger, Bradley and I, who worked together in the matter) that in these huge doses Myanesin *does* produce a state clinically resembling narcosis. Furthermore, Berger and Bradley (*op cit.*) state quite clearly that after doses of 50 mg. per kg. rabbits, though completely paralysed, "give the impression of being conscious" (*loc. cit.*).

Mr. Longley further states that cases of vasovagal collapse are "on record" in man but quotes no reference for his assertion. Myanesin was used in the human subject for various neurological conditions by two colleagues of mine, D. H. Belfrage (1947, 1947) and B. Buckley Sharp (1947) at my instigation. No reports of such a phenomenon have reached me from either of these workers, with one of whom at least I am in almost daily contact.

With regard to venous thrombosis: I found no greater incidence of this complication than following the administration of pentothal in the human subject, and Mr. Longley will find full details of my opinion, that intravenous *technique* is the answer to freedom from it, in my paper of 1948 (1948)).

Mr. Longley states that Myanesin has powerful cardio-inhibitory effects in rabbits in full dosage. I suggest that the dosage must be very full because Berger and Bradley (*op. cit.*) state that they observed no untoward effects on rabbits after doses as high as 100 mg. per kg., doses at least six times those in clinical use.

Lastly, Mr. Longley states that "Fatal post-anaesthetic nephritis" is recognised in human practice I contend that it would be fairer and more accurate to have stated that fatal renal damage following operation *has been suggested as being due* to the use of Myanesin. The lesions described were not nephritis but rather nephrotic. In my paper of 1948, on the other hand, Mr. Longley will find details of a patient who died following a severely traumatic and prolonged operation in which Myanesin was used, from kidney failure, together with a clear diagnosis of traumatic uraemia (also described as the crush syndrome) made from kidney sections I submitted to him, by Parmady whose brilliant work on this and allied conditions during and since the war is very well known. (1944, 1946 1947, 1947).

In conclusion, let me aver that I do not consider it is for me to put forward any opinions on the value of Myanesin in veterinary surgery, but having read the recent correspondence on the subject and influenced particularly by the letter of Joan O. Joshua (*Vet. Rec.* **63**, 810) may I put forward a plea for fair and unbiased statements and opinions on the subject backed by a thorough perusal of the literature dealing with both sides of the controversy and wherever possible backed further by statistical evidence based on a statistically significant number of carefully investigated cases?—Yours faithfully, F. Barnett Mallinson, Roseacre, Woldingham, Surrey. *December 3rd*, 1951.

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THE PRESIDENTIAL ADDRESS—SOME CRITICISM

Sir,—I was interested to read the report of Mr. Hodgman's Presidential address in to-day's issue of *The Veterinary Record*.

With many of his sentiments I am in full agreement, but to come straight to the point, I consider that his information, his logic or both, are at fault when he states that "a large number of practices are receiving a Governmental subsidy in the form of substantial L.V.I. fees."

Earlier the President states, "Change and growth are necessary conditions of life and our profession can look back on both. It has responded through the years to every changing need of the community; in the heyday of the horse it did great work for the nation's transport, and as the importance of the horse declined with rare elasticity it turned to the care of farm livestock and the smaller anima's." As he says, the services of the practitioner follow the needs and aims of the community at any given time and when these aims and needs are fair and legitimate so also are our efforts. Long before the administration of certain of the practitioners' services by the Ministry of Agriculture (commencing in 1938), it was evident in our district that farmers, in co-operation with their veterinary practitioners, were making determined efforts and much good progress in the control and eradication of all the diseases of our farm animals. I would not generalise as Mr. Hodgman has done, but I think it is unlikely that our local area was singular in this respect. Practitioners received and receive payment for doing their duty in connection with the above under a system of competition and strict professional discipline.

When part of their work is now administered by Government, why introduce the word "subsidy"? Mr. Hodgman, of course, does use the word "substantial" in relation to L.V.I. fees, but it may be of interest if I mention that personally, from my accountant's figures, I am unable to discover any clear relationship between changes in income and Ministry of Agriculture administration during the first five years of the change. After that other factors, such as the increased application of increased research in all veterinary problems, have to be considered carefully in assessing the sources of the practitioner's income. However, I am quite safe in stating that the practitioner has earned and will continue to earn his living as worthily as do the great majority of our fellow-countrymen.—Yours faithfully, A. J. Kennedy, Thornhill, Dumfriesshire. *December 1st*, 1951.

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THE VETERINARY SURGEON'S TITLE

Sir,—I have just received my copy of *The Veterinary Record* for November 3rd and would like to reply to the letter from Mr. W. T. Harrow that appears in that issue.

As he says, some years ago, during the war, there was some correspondence in your columns—in which I took part—about the title of "doctor" and the veterinary profession.

Here in Jamaica not only the Government veterinary surgeons but also the private practitioners are accorded the courtesy title, as also are a number of unqualified persons at present engaged in the treatment of animals. So far we are not protected by the Veterinary Surgeons Act and there is no way in which the qualified member can be distinguished from the "quack." While we welcome the courtesy title, we can only look forward to the day when legislation will be passed here, a supplementary register produced and the courtesy title withheld from our unqualified rivals.

In England, where members are now protected by the law and where such a supplementary register exists, it would be easy for a distinction to be made by giving to holders of a diploma or a degree the courtesy title of doctor. I do not agree with Mr. Harrow that the time is now opportune for such a change, I consider it long overdue.—Yours faithfully, Leonard R. Geffen, Kelroche, 99, Hope Road, Liguanea, Jamaica, B.W.I. *November 26th*, 1951.